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No. 5

CARCINOGENESIS ABSTRACTS

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Volume XIV, No. 5
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CARCINOGENESIS ABSTRACTS

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A monthly publication of the

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PREFACE

Carcinogenesis Abstracts is a publication of the National Cancer Institute. The journal serves as a vehicle through which current documentation of carcinogenesis research highlights are compiled, condensed, and disseminated on a regular basis. It represents an integral part of the Institute's program of fostering and supporting coordinated research into cancer etiology. Issues of *Carcinogenesis Abstracts* normally contain three-hundred abstracts and three-hundred citations (unaccompanied by corresponding abstracts). Abstracts and citations refer to the current scientific literature that describes the most significant carcinogenesis research carried on at the National Cancer Institute, other governmental agencies, and private institutions. *Carcinogenesis Abstracts* is intended to be a highly useful current awareness tool for scientists engaged in carcinogenesis research or related areas. The great number and diversity of publications relevant to carcinogenesis make imperative the availability of this service to investigators whose work requires that they keep abreast with current developments in the field.

Carcinogenesis Abstracts is normally published monthly. Volume XIV covers the scientific literature published from Jan 1976 through Dec 1976. To increase the usefulness of *Carcinogenesis Abstracts*, Volume XIV, a Wiswesser Line Notation index and a Chemical Abstracts Service Registry Number index have been provided. These indexes reference compounds described in abstracted articles. A cumulative subject, author, CAS Registry Number, and Wiswesser Line Notation index for Volume XIV will be published shortly after the final regular issue.

Carcinogenesis Abstracts is available free of charge to libraries and to individuals who have a professional interest in carcinogenesis. Requests for *Carcinogenesis Abstracts* from qualified individuals should include statements of their relationship to carcinogenesis research. All correspondence should be addressed as follows.

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The Secretary of Health, Education and Welfare has determined that the publication of this periodical is necessary in the transaction of the public business required by law of this Department. Use of funds for printing this periodical has been approved by the Director of the Office of Management and Budget through November 30, 1977.

NOTE

Journal names are abbreviated according to the list of abbreviations used by *Index Medicus*. For journals not covered by *Index Medicus*, the abbreviations found in *Chemical Abstracts Service Source Index*, 1907-1974 Cumulative, are used. New journals are verified in *New Serial Titles* and abbreviated according to *International Standard ISO 833*. An asterisk indicates the author to address (other than the primary) in requesting reprints.

LANGUAGE ABBREVIATIONS

Afr.	Afrikaans	Ind.	Indonesian
Ara.	Arabic	Ita.	Italian
Bul.	Bulgarian	Jpn.	Japanese
Chi.	Chinese	Kor.	Korean
Cro.	Croation	Lav.	Latvian
Cze.	Czech	Lit.	Lithuanian
Dan.	Danish	Nor.	Norwegian
Dut.	Dutch	Pol.	Polish
Eng.	English	Por.	Portuguese
Est.	Estonian	Rum.	Rumanian
Fin.	Finnish	Rus.	Russian
Fle.	Flemish	Ser.	Serbo-Croatian
Fre.	French	Slo.	Slovak
Geo.	Georgian	Spa.	Spanish
Ger.	German	Swe.	Swedish
Gre.	Greek	Tha.	Thai
Heb.	Hebrew	Tur.	Turkish
Hun.	Hungarian	Ukr.	Ukrainian
Ice.	Icelandic	Vie.	Vietnamese

ABBREVIATIONS USED IN ABSTRACTS

A	angstrom(s)	M	molar
ACTH	adrenocorticotropic hormone	mM	millimolar
ADP	adenosine diphosphate	µM	micromolar
AMP	adenosine monophosphate	mOsm	milliosmolar
ATP	adenosine triphosphate	mEq	milliequivalents
BCG	Bacillus Calmette Guerin	min	minute(s)
bid	twice daily	mo	month(s)
C	degrees centigrade	MTD	maximum tolerated dose
cal	calorie(s)	N	normal concentration
kcal	kilocalorie(s)	NAD	nicotinamide adenine dinucleotide
cc	cubic centimeter(s)	NADH	reduced nicotinamide adenine dinucleotide
Ci	curie(s)	NADP	nicotinamide adenine dinucleotidephosphate
mCi	millicurie(s)	NADPH	reduced nicotinamide adenine dinucleotide-phosphate
µCi	microcurie(s)		
cm	centimeter(s)	ng	nanogram(s) (10^{-9})
CNS	central nervous system	od	once daily
cpm	counts per minute	Pa	ambient pressure
dl	deciliter(s)	PAS	periodic acid-Schiff
ml	milliliter(s)	pg	picogram(s) (10^{-12})
µl	microliter(s)	pgEq	picogram equivalent
DNA	deoxyribonucleic acid	po	orally
ED$_{50}$	median effective dose	ppb	parts per billion
EDTA	ethylenediamine tetraacetic acid	ppm	parts per million
ESR	erythrocyte sedimentation rate	qid	four times daily
g	gram(s)	qod	every other day
kg	kilogram(s)	QO$_2$	oxygen quotient
mg	milligram(s)	R	roentgen(s)
µg	microgram(s)	RBC	red blood cells (erythrocytes)
Hb	hemoglobin	RNA	ribonucleic acid
hr	hour(s)	sc	subcutaneous
ia	intra-arterial	sec	second(s)
ic	intracerebral	SGOT	serum glutamic-oxalacetic transaminase
icav	intracavitary	SGPT	serum glutamic-pyruvic transaminase
id	intradermal	SRBS	sheep red blood cells
ILS	increased life span	TCD	tissue culture dose
im	intramuscular	TCD$_{50}$	median tissue culture dose
ip	intraperitoneal	tid	three times daily
ipl	intrapleural	U	unit(s)
it	intratumorous	mU	milliunit(s)
IU	International Unit	UV	ultraviolet
iv	intravenous	vol	volume
K$_m$	Michaelis constant	WBC	white blood cells (leukocytes)
LD	lethal dose	wk	week(s)
LD$_{50}$	median lethal dose	wt	weight
m	meter(s)	x	times
mm	millimeter(s)	yr	year(s)

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Veterinary Medicine

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2401 SELENIUM AND CANCER: A REVIEW. (Eng.) Schrauzer, G. N. (Univ. California at San Diego, Revelle Coll., La Jolla, CA 92093). *Bioinorg Chem* 5(3): 275-281; 1976.

The relation between selenium and cancer is reviewed. Selenium retards the growth of certain chemically induced tumors in animals. The addition of subtoxic amounts of this element in the form of selenite to the supply water lowers the incidence of spontaneous mammary tumors in female C_3H mice significantly without affecting the health and lifespan of the animals. Arsenic, a selenium antagonist, administered in the form of arsenite in the supply water, also lowers the tumor incidence of dosage levels of 10 ppm, but animals developing spontaneous mammary tumors under these conditions demonstrate dramatically enhanced tumor growth rates. The results of initial epidemiological studies suggest that human cancer mortality is lower in areas providing an adequate dietary intake of selenium, as estimated from the selenium content in grains and forage crops in various regions of the United States or the dietary selenium intakes calculated from food consumption data in various countries. (36 references)

2402 CAFFEINE, COFFEE, AND CANCER. (Eng.) Anonymous. (No affiliation given). *Br Med J* 1(6017): 1031-1032; 1976.

Caffeine remains a suspicious substance with respect to its carcinogenic effects despite both pro and con evidence. At extremely high levels, caffeine has been shown to be both teratogenic and mutagenic, but at lower levels, even prolonged studies have not shown any bad effects. There is even evidence of anticarcinogenic effects of caffeine in animals exposed to known carcinogens; it is also rapidly and efficiently metabolized in man. Even though there has been a correlation between caffeine consumption and renal and pelvic cancer in some studies, there is no convincing evidence to that effect. (21 references)

2403 THE NIH-SHIFT: ITS IMPACT ON THE MECHANISM OF AROMATIC HYDROXYLATION AND DRUG METABOLISM. (Eng.) Witkop, B. (Natl. Insts. Health, Bethesda, MD 20014). *Intra-Sci Chem Rep* 8(4): 75-87; 1974.

The chemistry of the NIH shift is reviewed. This phenomenon was discovered when tyrosine was formed from 4-^{3}H-phenylalanine by hydroxylating enzymes with retention of 95% tritium. Modified arene oxides not only cause but also alleviate cancer. The plant *Tripterygium wilfordi* contains the triepoxide tripolide, which has marked antileukemic properties. (55 references)

2404 PITFALLS IN EPIDEMIOLOGICAL RESEARCH: AN EXAMINATION OF THE ASBESTOS LITERATURE. (Eng.) Enterline, P. E. (Univ. Pittsburgh, Pittsburgh, PA 15261). *J Occup Med* 18(3): 150-157; 1976.

Estimates of the relative risks of death from respiratory cancer among workers occupationally exposed to asbestos compared to expected deaths calculated for various control groups ranged from 1.2 to 9.2 in 11 prospective studies in the U.S. and Canada undertaken between 1958 and 1972. This wide variation was due to the varying methods of analysis and selection of study and control groups by the different authors. The selection of appropriate latent periods has also differed, often because of the differing objectives of the studies. Some pitfalls include: correcting death certificates and then making comparisons with published vital statistics on uncorrected death certificates; deriving expected deaths from populations that do not reflect the mortality of the area in which the study population lives; failure to relate findings to exposure levels; overlapping exposure and followup periods in dose-response studies; and failure to adjust for competing causes of death. There is a need for further well-designed studies of asbestos-exposed workers to relate exposure and response more definitively. (38 references)

2405 STYRENE-LEUKEMIA LINK NOT FOUND IN STUDY. (Eng.) Anonymous (No affiliation given). *Chem Eng News* 54(18): 20-21; 1976.

Studies of the leukemogenic effects of polystyrene were conducted on animals and on workers in polystyrene plants in West Germany. A group of 40 rats was fed 5 g/kg polystyrene over 2 yr. The polystyrene contained about 0.1% residual styrene monomer. The diet had no effect on growth rate, weight increase, or lifespan, and there were no pathological differences in comparison to controls. A group of polystyrene workers were grouped according to age and duration of exposure. The usual air concentration of styrene in the plant was about 1 ppm. Comparison of these with 62 chloroquinone workers showed no significant differences in blood tests except for a better RBC count in the former. There was no evidence of liver damage in the polystyrene workers. In a U.S. study, 10 cases of leukemia were tentatively identified among styrene-butadiene workers. Other U.S. studies have also pointed to a possible correlation between leukemia and styrene-butadiene plants. (No references)

2406 NITRITE, NITROSAMINES, AND CANCER. (Eng.) Issenberg, P. (Univ. Nebraska Medical Center, Omaha, NB 68105). *Fed Proc* 35(6): 1322-1326; 1976.

Literature concerning nitrites and nitrosamines and their relationship to cancer is reviewed. Carcinogenic N-nitroso compounds are formed by unknown mechanisms, from the reaction of naturally occurring amines and nitrites that may be added to foods or produced by bacterial reduction of nitrate. N-Nitroso compounds can be produced during the processing, storage, and preparation of foods and in the mammalian stomach. Factors that influence nitrosation rates include pH, temperature, catalysts, and inhibitors. Predictions of the extent of nitrosation are complicated by these factors, and ultimately the amounts and types of N-nitroso compounds present must be determined by direct analysis. Methods to detect and estimate volatile nitrosamines are available, and low levels (parts per billion) have been found in some cured meat and fish products. General methods to detect all N-nitroso compounds are not available yet, but they are being developed. Evaluation of the risk to human populations from these compounds is difficult in the absence of more comprehensive data on their environmental distribution. (37 references)

2407 NERVOUS-SYSTEM-SPECIFICITY OF CARCINOGENESIS BY N-ETHYL-N-NITROSOUREA IN THE RAT: POSSIBLE SIGNIFICANCE OF O_6-GUANINE ALKYLATION AND DNA REPAIR. (Eng.) Rajewsky, M. F.; Goth, R. In: *Screening Tests in Chemical Carcinogenesis*. International Agency for Res. on Cancer. (Brussels, Belgium, 9-12 June 1975): no. 12, pp. 593-603; 1976.

A single pulse of the ethylating carcinogen N-ethyl-N-nitrosourea (ENU) applied to rats of perinatal age results in a high incidence of malignant neuroectodermal tumors associated with the central and peripheral nervous systems, after a dose-dependent induction period. This susceptibility correlates with a very slow removal of O^6-ethylquanine (O^6-EtGua) from the DNA of these tissues. The half-lives of ethylated purine bases in the DNA of different tissues after an ip pulse of ^{14}C-ENU to 10-day-old BD1X rats were: 229, 16, and 89 hr for O^6-EtGua, N^3-ethyladenine, and N^7-EtGua, respectively, in brain; and 36, 12, and 64 hr in liver. A similar selective persistence in the DNA of rat brain has also been demonstrated for O^6-methylguanine after the application of N-methyl-N-nitrosourea. With the high rate of DNA replication and cell division of the perinatal rat brain, the selective persistence of O^6-alkylated guanine in brain DNA may account for the increased probability of neoplastic transformation by the nitrosoureas. (27 references)

2408 REVIEW OF ANIMAL STUDIES. (Eng.) Williamson, K. S. (ICI Ltd., Central Medical Group, Fulshaw Hall, Wilmslow, Cheshire, SK9 1QB, England). *Proc R Soc Med* 69(4): 281-283; 1976.

Several studies are reviewed that have been conducted on animals exposed to vinyl chloride gas. It appears that the proportion of the dose retained and metabolized is greater following small doses than large ones. The amount that is inhaled is eliminated in three ways: unchanged from the lungs, in the carbon dioxide from the lungs, and by the excretion of metabolites in the urine. Although many studies are incomplete, there is substantial evidence to indicate that vinyl chloride is oncogenic. (9 references)

2409 MOLECULAR AND CELLULAR MECHANISMS OF CHEMICAL CARCINOGENESIS. (Eng.) Weinstein, B. In: *Cancer Biology. II. Induction, Regulation, Immunology and Therapy. Based on a Series of Lectures Pre-Aspen, Colorado, July 1974.* Edited by R. R. Pascal, F. Silva and D. W. King. (New York: Stratton Intercontinental Medical Book Corp.): pp. 4-9; 1976.

Transformation of adult rat liver by the N-acetoxy derivative of N-2-acetylaminofluorene (AAF), as a model of chemical carcinogenesis, is reviewed. These cells have an epithelial morphology and stop dividing when the culture reaches a confluent monolayer. AAF if activated to an electrophile in the liver can attach to the C-8 position of any guanine-containing nucleic acid, probably by displacing the guanine from its normal position in the helix. This could lead to a frame shift mutation during DNA replication, which might occur during carcinogenesis if the somatic mutation hypothesis is correct. Base displacement might be the mechanism of other planar chemical carcinogens, such as the polycyclic hydrocarbons. Mutants of chemically transformed epithelial cells which are temperature-sensitive (TS) in the maintenance of transformation were investigated. The TS mutation appears to specifically affect the saturation density of the cells and their ability to grow in agar. Scanning electron microscopy showed that at 40 C the cells were flat and smooth, while at 36 C there were patches of piled-up spherical cells with a rough surface. Cultures established from hepatomas induced in the rat with aromatic amine carcinogens contained large numbers of budding C-type particles and intracytoplasmic A-type particles. These viral particles are believed to be symptomatic of defects in the control of gene expression occurring during chemical carcinogenesis that are associated with derepression of latent viruses. No biologic action of these viral particles has been found. (5 references)

2410 EFFECTS OF DIETARY CONSTITUENTS ON THE METABOLISM OF CHEMICAL CARCINOGENS. (Eng.) Wattenberg, L. W. (Univ. Minnesota Sch. Medicine, Minneapolis, MN 55455). *Cancer Res* 35(11/Part 2): 3326-3331; 1975.

Two types of dietary constituents have been shown to affect the metabolism of chemical carcinogens by the microsomal mixed-function oxidase system. Naturally occurring inducers of increased activity of this system are present in plants. Cruciferous vegetables (including Brussels sprouts, cabbage, and cauliflower) are relatively potent in this regard. From these vegetables, three indoles with inducing activity have been identified. These are indole-3-acetonitrile, indole-3-carbinol, and 3,3'-diindolylmethane. A second type of dietary constituent affecting the microsomal mixed-function oxidase system is added phenolic antioxidant, namely butylated hydroxyanisole (BHA) and butylated hydroxytoluene. Studies of the effect of BHA on metabolism of benzo(a)pyrene by liver microsomes have been carried out. BHA feeding results in microsomal changes. The cytochrome P-450 shows altered spectral characteristics; the aryl hydrocarbon hydroxylase system of these microsomes has an increased sensitivity to inhibition by alpha-naphthoflavone. Furthermore, there is a decrease in binding of metabolites of benzo(a)pyrene to DNA upon incubation of these microsomes with benzo(a)pyrene. Experimental studies of induction of increased mixed function oxidase activity have shown that increased levels of activity protect against administration of chemical carcinogens. BHA and butylated hydroxytoluene have been found to exert a protective effect against chemical carcinogens. (58 references)

2411 A HAZARDOUS SOCIETY? INDIVIDUAL VERSUS COMMUNITY RESPONSIBILITY IN CANCER PREVENTION. THIRD ANNUAL MATTHEW B. ROSENHAUS LECTURE. (Eng.) Higginson, J. (International Agency Res. Cancer, 150 Cours Albert Thomas, 69008 Lyon, France). *Am J Public Health* 66(4): 359-366; 1976.

Environmental and occupational hazards associated with cancer are reviewed, with special emphasis on the role of the individual and that of the community in primary cancer prevention and control. The concept of chemical environment is developed, and the importance of environmental factors in human cancer is summarized. The cancer patterns of today reflect stimuli in the environment 25-50 yr ago. The concept of risk is considered in terms of age, personal habits, and relative importance of cancers at different relative risks in the general population. For most of the carcinogens present in the environment, adequate epidemiological data are not available to calculate actuarial risks for man at present exposure levels. The concept of socially acceptable risk is discussed, and the role of common sense and honesty in accepting such a concept is stressed. The adoption of a personal cancer plan through modification of one's lifestyle is strongly recommended. (35 references)

2412 STEROID HORMONE REGULATION OF GENE EXPRESSION. (Eng.) O'Malley, B. W. In: *Cancer Biology. I. Induction, Regulation, Immunology and Therapy. Based on a Series of Lectures Presented at the Given Institute of Pathobiology in Aspen, Colorado, July 1974.* Edited by R. R. Pascal, F. Silva and D. W. King. (New York: Stratton Intercontinental Medical Book Corp.): pp. 34-37; 1976.

The effects of steroid hormones on the regulation of gene expression with the chick oviduct as a model, are reviewed. A steroid hormone receptor in chick oviduct cells has been purified to homogeneity and characterized as having two protein molecules, each of which possesses a steroid binding site and one of which also possesses a DNA binding site. Chick oviduct shows specific responses to estrogen and progesterone. In the newly hatched chick, oviduct growth and development is dependent upon estrogen, as is the synthesis of ovalbumin. Progesterone produces no growth changes, but is required for the synthesis of avidin. The administration of diethylstilbestrol (DES) to chicks yields both increased intracellular ovalbumin and increased mRNA activity. The increased mRNA activity correlates with the rate of ovalbumin synthesis. Progesterone produces a concurrent increase in mRNA activity and rate of synthesis of avidin. Thus, steroid hormones influence mRNA in their action of cells. Ovalbumin mRNA can be purified 97.5% compared to a whole cell extract, and codes specifically for ovalbumin synthesis. It appears that one gene is responsible for the production of relatively large amounts of specific ovalbumin mRNA. Template activity assays before and after hormone stimulation indicate that ''chromosomal proteins maintain binding sites in unstable forms.'' With DES stimulation of the chromatin, the ability of the template to bind and stack RNA polymerase increases rapidly. The main effect of steroid hormone-receptor complexes appears to be stimulation of transcription at the level of initiation. These findings suggest that some hormones may disrupt normal DNA molecules in such a way as to enhance development of neoplasia. (8 references)

2413 SOMATIC EFFECTS OF SMALL DOSES OF IONIZING RADIATION IN HUMANS. (Fre.) Goutier, R. (Laboratoire de Radiobiologie, 149 boulevard de la Constitution, 4020 Liege, Belgium). *J Belge Radiol* 59(1): 21-33; 1976.

A chapter from the 1972 report on the Biological Effects of Ionizing Radiation (prepared by the U.S. Academy of Sciences) dealing with somatic effects in man is analyzed. The conclusions reached by the committee on the carcinogenic risk of low-level radiation are disputed because they are based on statistics extrapolated from studies on humans exposed to irradiation at relatively high dose levels and very high dose rates. The studies include those on survivors of Nagasaki and Hiroshima, large series of patients treated with radiotherapy for ankylosing spondylitis and menstrual disorders, uranium miners, and persons professionally or therapeutically exposed to high doses of radium radioisotopes. The extrapolation procedure is reliable if three conditions of radiation are met: (a) the dose-effect relationship is linear throughout the entire dose range of radiation; (b) a threshold dose of radiation does not exist; that is, a dose below which no effect is observable; (c) intensity of effect is independent of dose rate. Based on a new analysis of the studies, it is concluded that these conditions are met by the highly ionizing radiation of α rays and neutrons but not by x- and γ rays. Extrapolated values, in a table reproduced from the report, on the latent period of cancers, the plateau (high-risk) period, and the absolute and relative risk expressed per rem per 10^6 persons per year for leukemia (having the highest risk) and other cancers are considered valid only if applied to α rays and neutrons. (19 references)

2414 THE USE OF IN VITRO METHODS FOR THE STUDY OF X-RAY-INDUCED TRANSFORMATION. (Eng.) Klein, J. C. In: *Biology of Radiation Carcinogenesis.* Edited by J. M. Yuhas; R. W. Tennant; J. D. Regan. (New York: Raven Press): pp. 301-307; 1976.

Cell culture systems can be used to investigate malignant transformation directly at the cellular level with the exclusion of interfering factors that operate in whole animals. Chemical, viral, and radiation carcinogenesis can be investigated in the same system. Transformation may be assessed either by in vivo tests or by the in vitro cloning technique whereby transformed piled-up colonies are distinguished from nonmalignant colonies and scored. One pitfall in in vitro studies is that many cells may be resistant to transformation by irradiation. Cells from two of three sublines of a cloned mouse spleen cell line were malignantly transformed (as demonstrated by in vivo tests) by irradiation with 300 rads of x-rays (this dose killing 80% of the cells). A control experiment was designed to test for the effects of high cell density (10^6cells/ml) and for the presence of damaged cells: 80% of the cell suspension was removed, the cells were killed by repeated freezing and thawing, and then the original suspension was reconstituted by returning the killed cells to the residual viable portion. The sublines that were transformed by irradiation were also transformed without irradiation merely by killing part of the cell population in this way. It therefore appears that material from damaged normal cells can cause malignant transformation. (23 references)

2415 EXPERIMENTAL PATHOLOGY OF AGING. (Eng.) Tuchweber, B. (Institut de Medecine et de chirurgie experimentales, Universite de Montreal, Montreal, Quebec, Canada); Salas, M. *Methods Achiev Exp Pathol* 7: 167-226; 1976.

The following topics are reviewed: (a) the effects of normal aging on the heart, blood vessels, kidney, bone, cartilage, nerves, muscle, endocrine organs, thymicolymphatic and immune systems, and on other tissues; (b) factors that modify normal aging, such as stress, hormone balance, diet, and drugs; (c) cellular aging characteristics such as the accumulation of age-related pigments; (d) induced premature aging in animals; and (e) possible mechanisms of aging, such as the cumulative effect of mutations in somatic cells and the functional decline in the immune system. A number of phenomena characteristic of senility, for instance, changes in the physical properties and chemical composition of collagen and elastin, may be ascribed to immobilization of macromolecules by cross-linking. Restriction of dietary intake prolongs the life span, probably by depressing anterior pituitary gland function. (296 references)

2416 XERODERMA PIGMENTOSUM, DNA REPAIR AND CARCINOGENESIS. (Eng.) Cleaver, J. E. In: *Cancer Genetics*. Edited by H. T. Lynch. (Springfield, IL: Charles C. Thomas): pp. 111-121; 1976.

Xeroderma pigmentosum (XP) is a rare human disease, normally inherited as an autosomal recessive gene, in which the skin is extremely sensitive to the short wavelength region of the sun's spectrum. Sunlight produces skin cancers of both ectodermal and mesodermal origin (basal cell carcinomas, squamous cell carcinomas, melanomas, angiosarcomas, fibrosarcomas, and keratoanthomas). Fibroblasts from XP patients are in general much more sensitive in vitro to the lethal effects of UV irradiation than normal cells, and this has been shown to result from a diminished capacity for repairing UV-damaged DNA. Most fibroblasts from donors in North America have a residual repair capacity that is below 55% of normal; higher levels have been found in some Dutch cases, in which the severity of the disease has been correlated to reduction in repair capacity. Most heterozygotes demonstrate normal repair levels. The observation of at least four mutually complementary genetic defects in XP implies that the defective DNA repair system may be one multiunit enzyme with subunits that have different functions, or that there are several different enzymes involved. Three possible, and not necessarily mutually exclusive, mechanisms are suggested for the occurrence of cancer in XP patients: (a) an increased mutation rate; (b) the induction of oncogenic viruses by low UV doses; and (c) an increased malignant transformation of UV-irradiated cells by oncogenic viruses. (39 references)

2417 REVERSE TRANSCRIPTASE. (Eng.) Wu, A. M. (Litton Bionetics, Bethesda, MD); Gallo, R. C. *CRC Crit Rev Biochem* 3(3): 289-347; 1975.

A very comprehensive review of the biochemical, biophysical, and immunological properties of reverse transcriptase is presented. The enzyme has been used to help identify and quantify viruses, to prepare molecular complementary DNA probes, to search for signs of viruses in tissues not known to be producing viruses, and to provide information about virus replication. Whereas the origin and role of the enzyme in virus-infected cells are clear, the possible role of ''intrinsic'' cellular reverse transcriptase involving gene modification or amplification in development and differentiation is more speculative. At present there is no definitive evidence that the normal adult tissues of higher organisms contain authentic reverse transcriptase, although some observations have suggested this possibility. There is some evidence indicating that intrinsic enzyme is present in embryonic tissues. Extrinsic viral enzyme, besides synthesizing provirus, may cause abnormal cell proliferation either by affecting gene modification or amplification or by integrating provirus into the host chromosomes at some specific location. (437 references)

2418 REVERSE TRANSCRIPTASE AND POSSIBLE HUMAN TUMOUR VIRUSES. (Eng.) Michalides, R. (Netherlands Cancer Inst., Amsterdam, The Netherlands). *Int Union Cancer Tech Rep Ser* 20: 114-118; 1975.

The mechanism of action and properties of RNA reverse transcriptase of RNA tumor viruses are reviewed in an effort to deduce information about a possible human RNA tumor virus. The action of the reverse transcriptase of RNA tumor viruses can be shown in both endogenous (within the RNA tumor virus) and exogenous reactions. In either type of reaction using the viral 60-70S RNA, the newly synthesized DNA is covalently linked to a small RNA molecule. This small RNA appears to have the properties of cellular tRNA and is necessary for the action of the reverse transcriptase. In both endogenous and exogenous reactions using the 60-70S RNA, the average length of the DNA which is transcribed from the 60-70S RNA is only 130 nucleotides, while in vivo a total DNA transcript has been found. This suggests the existence of elongation factors that are inactive in in vitro assays. The simultaneous detection test, which reveals the presence of 60-70S RNA simultaneously with reverse transcriptase, has shown viruses in human milk, breast cancer, leukemia, and sarcomas, including Hodgkin's lymphoma. Molecular hybridization using DNA transcripts from the 60-70S RNA of known RNA tumor viruses has shown a certain degree of homology between the RNAs from human tumors and animal tumor viruses. An enzyme has been isolated and characterized from human leukemic cells with properties of the reverse transcriptase from RNA tumor viruses. Particles with the properties of RNA tumor viruses have been isolated from human leukemic cells in tissue culture. The viral etiology of cancer in animals suggests that genetic information from oncogenic RNA viruses could be involved in human cancer. (4 references)

2419 THE MAMMARY TUMOUR VIRUSES. (Eng.) Hageman, P. C. (No affiliation given). *Int Union Cancer Tech Rep Ser* 20: 98-105; 1975.

The isolation and characterization of mouse mammary tumor viruses (MTV) including descriptions of some analytical techniques, are discussed. Various MTV strains have been isolated from different mouse strains; the exogenous MTV, strains are transmitted by infection, mostly by the mother's milk, while the endogenous MTV strains are genetically transmitted and are usually only slightly oncogenic. The MTV virion is a B particle and contains reverse transcriptase in its core. The major polypeptides are p 28, p 14, and p 10, and the glycoproteins, gp 52 and gp 36. Information for MTV appears to be present in the DNA of all mouse strains investigated. Transcription to RNA occurs in many more tissues than expected, but formation of mature virions is limited to the epithelial cells of the mammary gland and the epididymis. The mouse MTV is the only MTV proven to cause mammary tumors. Indications for the presence of an oncogenic RNA (oncorna) virus are found in human milk and mammary tumors, but the viral etiology of the tumors is not established. MTV can be purified from mouse milk of high mammary-tumor strains and from mammary tumors of these strains but the best starting material is medium from MTV-producing tissue cultures. The method of choice of purification for all oncornaviruses is centrifugation in density gradients. Determination of the infectivity can only be performed in mice of a susceptible strain; titration in vitro in tissue culture is not yet possible. Other detection techniques include electron microscopy, which can establish the presence of mature virions, immunological methods to detect viral antigens, reverse transcriptase tests, and molecular hybridization. Procedures are included for the isolation of B (or C) particles from tumor tissue, extraction of MTV antigens from tissue, preparation of anti-MTV serum in rabbits, absorption of aspecific antibodies from anti-MTV serum, and preparation of micro-Ouchterlony slides. (23 references)

2420 ONCOGENIC RNA VIRUSES. (Eng.) Gallo, R. C. (Natl. Cancer Inst., Natl. Insts. Health, Bethesda, MD 20014); Todaro, G. J. *Semin Oncol* 3(1): 81-95; 1976.

The transmission of RNA tumor viruses in vivo, characteristics of their growth in vitro, the isolation of type C viruses from primates, and the possible association of RNA tumor viruses with human cancer are reviewed. Transmission of the viral species can be vertical, from parent to progeny, horizontal, by infection, or congenital, from mother to fetus via an infective mechanism. Type C RNA viruses have been isolated from many vertebrate species and they have been demonstrated to cause a variety of naturally occurring vertebrate cancers. Complete infectious type C viruses have recently been isolated from human material. The best-characterized isolate is HL-23, from a cell culture derived from a woman with acute myelogenous leukemia. Various lines of investigation (detection of viral reverse transcriptase and molecular hybridization) are being pursued to determine if viral information is involved in human neoplasia. Although the majority of human cancers are not caused by virus by a classical route, viral information may be involved in many or all cancers in a complex manner that comprises both genetic and environmental factors. Many properties of viral RNA are more like those of the heterogenous nuclear RNA of mammalian cell nuclei than those of mature messenger RNA, indicating that viral RNA may not be processed identically to the majority of cell RNA. Perhaps means may be found to interfere with the maturation of viral RNA without affecting the bulk of cell RNA. (73 references)

2421 ONCOGENIC DNA VIRUSES--REPLICATION, TUMOR GENE EXPRESSION, AND ROLE IN HUMAN CANCER. (Eng.) Green, M. (St. Louis Univ. Sch. Medicine, 3681 Park Ave., St. Louis, MO 63110 ; Wold, W. S. M. *Semin Oncol* 3(1): 65-79; 1976.

A review is presented of the origin and properties of DNA-containing papovaviruses, adenoviruses, and herpesviruses. Molecular events in the replication of viruses during productive infection and cell transformation in appropriate hosts are discussed. The main event in transformation is a change in cell growth regulation brought about by the functions of a small number of integrated transforming viral genes. The adenoviruses provide extremely simple one- or two-gene systems for studying transformation. Oncogenic viruses may have a role in initiating human cancers. Very highly radiolabeled adenovirus DNA molecules representing either the entire genome or selected portions are being used to test, by molecular hybridization, whether tumor cell or normal cell DNA can contain integrated copies of viral DNA. As few as 0.1 copy of viral DNA per cell can be detected. DNA sequences related to adenovirus 2 have been detected in several human tumors, but it is not yet certain that these sequences represent transforming genes that initiated the tumors. (31 references)

2422 FROM THE MOLECULAR BIOLOGY OF ONCOGENIC DNA VIRUSES TO CANCER. (Eng.) Dulbecco, R. (Imperial Cancer Res. Fund Labs., Lincoln's Inn Fields, London, W.C.2, England). *Science* 192(4238): 437-440; 1976.

The relationship between oncogenic DNA viruses and cancer is reviewed. Early studies showed that polyoma virus could be assayed in certain cell cultures (permissive) and could induce a cancerlike state in other cultures (nonpermissive), this induction being called transformation. Integration is one of the key events in virus-induced cell transformation. Cell genes play a continuing role in transformation, but the relationship between viral gene expression and cell phenotype is complex. Further progress came from studies of the proteins, the T-antigens in particular, specified by the early region of the viral DNA. Since the early, transforming part of the SV40 genome can specify proteins of a molecular weight of about 100,000 daltons, the T antigen is likely to be its sole product and, therefore, the transforming protein. Cellular events during transformation include functional changes and mutations, which are not well understood. Further knowledge of the mechanisms of growth regulation in animal cells is also necessary. The possibility that the viral transforming protein replaces one of the normal nuclear regulatory proteins of the cell is considered. For control of cancer it would be important to recognize the developmental period in which the anachronistic genes of transformed or cancer cells are normally expressed. Because of the persistence of viral DNA in the transformed cells, whether or not a given DNA is a possible agent of human cancer might be ascertained by looking for its DNA in the cancer cells. The feasibility of cancer prevention is indicated by the fact that promutagens already identified in preliminary screening (e.g., tobacco, some hair dyes) are inessential for human life. (33 references)

2423 VIRUSES, POLYMERASES, AND CANCER.

(Eng.) Baltimore, D. (Dept. Microbiology, Massachusetts Inst. Technology, Cambridge, MA 02139). *Science* 192(4240): 632-636; 1976.

In this Nobel Prize lecture, work on the study of different genetic systems of RNA viruses is reviewed. Early studies were concerned with picornavirus RNA production and the various enzymes involved, with poliovirus being the principal subject. Only three species of RNA are seen in poliovirus-infected cells, but with vesicular stomatitis virus (VSV), at least nine RNA's are produced, one of which is heterogeneous. The discovery of a virion polymerase in VSV led to the search for these enzymes in other viruses, particularly RNA tumor viruses. Two distinguishable time periods follow infection of a cell by a retrovirus: a period of reverse transcription and establishment of the DNA provirus as an integrated part of cellular DNA, and a period of expression of the integrated genome and synthesis of progeny virions. The biochemical events of each of these periods are summarized. Three kinds of explanations have been proposed for virus-related genes that are inherited in the germ line of so many animal species. The first is that they are an aspect of the animal and are virus-related because they are the progenitors of retroviruses. The second suggests that they are genes inserted into the chromosome of some ancestral animal by retrovirus infection of the germ cells of that animal. The third explanation, considered the most likely, is a modification of the second, in which any positive role for viral genes in the life of the infected animal is excluded. (62 references)

2424 THE ROLE OF C-TYPE AND OTHER ONCOGENIC VIRUS PARTICLES IN CANCER AND LEUKEMIA

(Editorial). (Eng.) Gross, L. (Veterans Admin. Hosp., Bronx, NY 10468). *N Engl J Med* 294(13): 724-725; 1976.

Information concerning the role of type C and other oncogenic virus particles in cancer and leukemia is reviewed. A, B, and C particles are usually spherical, with two or more concentric shells, and are found within or outside the cells. Type B particles are present in mammary cancer in several mouse strains and in a rhesus monkey mammary tumor. Type C particles are found in primary or experimental virus-induced lymphosarcomas, sarcomas, and fibrosarcomas of mice, cats, chickens, and woolly monkeys and in primary lymphosarcomas in baboons and gibbon monkeys. Virus particles have not been detected in primary human lymphosarcomas or carcinomas. Even sarcomas in hamsters or rats induced with tissue culture fluids containing innumerable virus particles do not reveal the presence of these particles. (14 references)

2425 THE CHROMOSOMAL LOCALIZATION OF AN ENDOGENOUS MURINE LEUKEMIA VIRAL GENOME IN THE AKR MOUSE.

(Eng.) Lowy, D. R.; Chattopadhyay, S. K.; Teich, N. In: *Biology of Radiation Carcinogenesis*. Edited by J. M. Yuhas, R. W. Tennant, and J. D. Regan. (New York: Raven Press): pp. 195-205; 1976.

Literature concerning the location in the AKR mouse of the murine leukemia virus (MLV) genome, which indicates that it is part of the normal chromosomal genetic material of AKR cells, is reviewed. Within 1 wk of postnatal life, the high leukemic AKR mouse becomes positive for a mouse-tropic, C type RNA virus. Several copies of this viral genome are present in AKR cellular DNA, as indicated by nucleic hybridization studies utilizing a single-strand DNA probe complementary to the RNA of this virus. Breeding experiments have shown that the expression of this virus is determined by classic Mendelian segregation: the chromosomal location of a virus-inducing locus (*Akv-1*) has been mapped. Combined biologic and biochemical studies demonstrate that the *Akv-1* gene contains viral sequences. Additional studies suggest that C type viral genetic information is present in the chromosomal DNA of many mouse strains. The two types of cellular control of viral replication are transcriptional control of endogenous viral DNA and cellular susceptibility to exogenous infection by endogenously activated virus. These results do not exclude the possibility of infection by C type viruses from outside the organism; they do, however, provide some understanding of the epidemiological observation that, in mice, the C virus is usually transmitted vertically. (36 references)

2426 INFECTIOUS PRIMATE TYPE C VIRUS GROUP: EVIDENCE FOR AN ORIGIN FROM AN ENDOGENOUS VIRUS OF THE RODENT *MUS CAROLI*. (Eng.) Todaro, G. J. (Natl. Cancer Inst., Natl. Insts. Health, Bethesda, MD 20014); Benveniste, R. E.; Sherr, C. J.; Lieber, M. M.; Callahan, R. *Bibl Haematol (Basel)* (43): 115-120; 1976.

Gene sequences homologous to those of the RNA's of GALV (gibbon ape leukemia virus) and SSAV (simian sarcoma-associated virus) have not been detected in the DNA of normal primates studied to date, and the transmission of these viruses appears to be horizontal. With nucleic acid hybridization techniques, it was observed that GALV and SSAV share partial sequence homology with endogenous type C viruses from the laboratory mouse, which suggests that this group of viruses may have originated by a transspecies infection. A type C virus isolated from the Thai mouse *Mus caroli* is closely related antigenically to the gibbon and woolly monkey type C viruses and only weakly related to any of the previously described mouse type C viruses. It shares cross-reactive reverse transcriptase and p30 antigens, and it cross-interferes with the infectious primate type C viruses. The apparent cross-species infection appears to have been a relatively recent event, with the virus not yet being incorporated into the genomes of the recipient primate species. The possible association of type C primate viruses with human acute leukemia derives from several observations. An enzyme with biochemical properties related to those of type C viruses and with antigenic properties similar to polymerases of the woolly monkey type C virus and the gibbon ape leukemia virus has been detected in human acute leukemia cells. Antigens related to the major structural proteins, p30, of type C viruses have been detected in the peripheral WBC of five patients with acute leukemia. Complete infectious type C viruses have been recently isolated from various human materials, but their ultimate significance is not as yet known. (40 references)

2427 NONHUMAN PRIMATE ONCOGENIC HERPESVIRUSES: VIRUS-HOST RELATIONSHIPS. (Eng.) Laufs, R. (Hygiene-Institut der Universitaet, D-34, Goettingen, W. Germany). *Bibl Haematol (Basel)* (43): 331-335; 1976.

Malignant tumors in a number of nonhuman primate species resulting from inoculation with isolates of Herpesvirus saimiri (HVS) and Herpesvirus ateles (HVA) are reviewed and their relevance to human carcinogenesis is discussed. Both HVS and HVA induce a latent infection in their natural hosts, squirrel monkeys and spider monkeys, respectively, but not a malignant disease. However, both cause malignant, fatal diseases after they are injected into several closely related primate species. Koch's postulates are fulfilled for these oncogenic effects. There is no evidence for an interaction between the oncogenic simian herpesviruses and type C viruses, which provides indirect support for the hypothesis that Epstein-Barr virus may contribute to the etiology of Burkitt's lymphoma and nasopharyngeal carcinoma in man. Studies of humoral immune responses to HVS have defined a number of immune parameters that appear to reflect the course of the virus infection, but cellular immune responses in resistance to HVS remain to be defined. Transformation of marmoset and human lymphocytes in vitro with HVA has made it possible to establish T and B lymphocyte cell cultures from the same monkey or human donor and to compare their membrane antigens and functional properties. Both HVS and HVA are good models for herpesvirus vaccine studies, since they induce tumors in animals phylogenetically related to man and since the biological aspects of the virus-host interactions have been well-defined. (33 references)

2428 EPIDEMIOLOGY OF THE EPSTEIN-BARR VIRUS INFECTION AND ASSOCIATED TUMORS IN MAN. A TOOL FOR ETIOLOGY AND CONTROL. (Eng.) de The, G. (International Agency Res. Cancer, 150, cours A.-Thomas, F-69008, Lyon, France); Day, N.; Geser, A.; Ho, J. H. C.; Simons, M. J.; Sohier, R.; Tukei, P.; Vonka, V. *Bibl Haematol (Basel)* (43): 216-220; 1976.

The epidemiological characteristics of Epstein-Barr virus (EBV) in relation to Burkitt's lymphoma (BL), nasopharyngeal carcinoma (NPC), and infectious mononucleosis (IM) are reviewed. The geographical distribution of these three EBV-associated diseases differs considerably, and, to a great extent, they are mutually exclusive worldwide. IM prevails in upper socioeconomic groups in temperate zones, while BL incidence exhibits time/space clustering and seasonal variations that suggest the intervention of a triggering environmental factor, relatively close in time to the onset of the disease. The risk for NPC appears to be linked with genetic and cultural factors. Whether the mode of transmission of EBV is the same in tropical, subtropical, and temperate zones is unknown. The immune response to EBV antigens is different among different ethnic groups. It has been suggested that very early EBV infection, possibly in the perinatal period, is an important if not crucial risk factor for BL. The IARC (International Agency for Research on Cancer) study on BL in the West Nile District of Uganda is described briefly, and although it is too early to draw definite conclusions, it appears that BL is caused by a chronic and heavy EBV infection that results in high EBV antibodies long before the onset of disease. Genetic factors appear to be involved in the different age distribution of NPC among Chinese and Africans. The possible control of EBV-associated diseases is discussed. (22 references)

2429 SERUM IgA ANTIBODIES TO EPSTEIN-BARR VIRUS (EBV)-RELATED ANTIGENS, NEW FEATURE OF NASOPHARYNGEAL CARCINOMA. (Eng.) Henle, G. (Children's Hosp. Philadelphia and Sch. Medicine, Univ. Pennsylvania, 34th St. and Civic Center Blvd., Philadelphia, PA 19104); Henle, W. *Bibl Haematol (Basel)* (43): 322-325; 1976.

The key features of Epstein-Barr virus (EBV)-related serology in infectious mononucleosis, Burkitt's lymphoma, and nasopharyngeal carcinoma are reviewed, with special emphasis on the level of IgA antibody to viral capsid antigen (VCA). Nearly all patients with untreated nasopharyngeal carcinoma have serum IgA to VCA, and many have antibodies to the diffuse component of EBV-induced early antigens, often at high titers that occasionally equal the corresponding IgG titers. Patients with other head and neck tumors rarely showed EBV-specific IgA, and only 28% of Burkitt's lymphoma patients have low levels of VCA-specific IgA. No more than 40% of infectious mononucleosis patients have weak, transient VCA-specific IgA. Nasopharyngeal carcinoma patients examined 2-6 yr after treatment have little or no VCA-specific IgA, suggesting a possible role for this antibody in followup. Possible explanations for this unique incidence and often high titer of EBV-specific IgA antibody in untreated patients with nasopharyngeal carcinoma are discussed. (17 references)

2430 HUMAN CANCER VIRUSES. (Eng.) Miller, G. (Yale Univ. Sch. Medicine, New Haven, CT). *Adv Intern Med* 21: 189-219; 1976.

The association of viruses with human tumors is discussed with respect to methodological approaches and specific examples of virus-associated tumors. Major methods used to detect viral genomes in tumors include molecular hybridization with nucleic acids and the detection of viral-specified proteins. To establish that a virus is the etiologic agent for a human cancer, one must follow the Koch-Henle postulates: the organism must be readily isolated from patients with the illness; the virus must be isolated in pure state; and experimental disease must be induced with the virus in question. Because of various difficulties involved in determining accordance with any of these postulates, no single category of evidence can be considered sufficient proof that a human virus causes cancer. Other types of evidence that taken together might contribute such proof include: demonstration of the virus in the tumor, recovery of virus from tumors in vitro, cell transformation in vitro, experimental tumorigenesis in animal, seroepidemiologic association, and epidemiologic proofs. The most persuasive evidence for the existence of a human tumor virus comes from studies of the Epstein-Barr virus (EBV), which is intimately associated with infectious mononucleosis, Burkitt's lymphoma (BL), and nasopharyngeal carcinoma (NPC). In general, the data are highly suggestive of an oncogenic role for EBV in the latter two. Carcinoma of the cervix appears to be associated with Herpes Simplex Type 2, the biologic properties of which are compatible with properties anticipated for an oncogenic virus. Experiments attempting to relate an agent similar tc the murine mammary tumor virus to human breast cancer have also provided suggestive but incomplete evidence for an etiologic association, and the role of herpesviruses in leukemogenesis in man should be evaluated. At least three different classes of viruses found in man possess biologic attributes of tumor viruses: adenoviruses, cytomegaloviruses, and papovaviruses. Implications of the possible viral etiology of human cancer for prophylaxis are discussed. (97 references)

2431 VIRUSES AS AN ETIOLOGIC FACTOR IN CANCER. (Eng.) Rapp, F. (Milton S. Hershey Medical Center, Pennsylvania State Univ. Coll. Medicine, Hershey, PA 17033). *Semin Oncol* 3(1): 49-63; 1976.

The properties of oncogenic papovaviruses, herpesviruses, and RNA-containing viruses are reviewed with particular emphasis on the experimental induction of tumors in animals and on the possible involvement of these viruses in the etiology of human cancers. Evidence for the existence of viral information in human cancers can now be sought by a number of different techniques. It may be more profitable to look for gene products, such as antigens, reverse transcriptases, and core proteins, rather than to look directly for the viral nucleic acids by hybridization experiments, because in the former cases amplification of the material has occurred via transcription and translation. (51 references)

2432 MEMBRANE CHANGES IN VIRUS TRANSFORMED CELLS. (Eng.) Montagnier, L. (Pasteur Inst., Paris, France); Torpier, G. *Bull Cancer (Paris)* 63(1): 123-134; 1976.

Membrane changes associated with viral transformation are reviewed, particularly those in the hydrophobic part of the plasma membrane as visualized by freeze fracture electron microscopy. Viral transformation brings about: an increased exposure of surface glycolipids accompanied by a decrease in the content of fully glycosylated species; an increase in the glycosylation of glycoproteins; a partial disorganization of underlying microtubules caused by decreased cyclic AMP levels, which perhaps causes a loss of cell anchorage dependence; loss or gain of particular surface glycoproteins; and an increase in the number of intramembranous particles (IMP) on the freeze-fractured A face (the outwardly directed face of the inner phospholipid leaflet). The density of distribution of the A face IMP of untransformed BHK21/13/8 cells was 552 particles/ μm^2. After transformation by hamster sarcoma virus (an RNA virus), the density was increased to 1.673 particles/μm^2, and after transformation by polyoma virus (a DNA virus), it was 1,069 particles/μm^2. Results with cells that are temperature-sensitive for transformation have suggested that the majority of these membrane changes must be dependent on the expression of a single viral gene or, at most, several genes. Some changes must therefore be produced secondarily, as a consequence of others. It is suggested that the primary change induced by the transforming viruses is associated with the hydrophobic part of the membrane. (33 references)

2433 HOST IMMUNE RESPONSE TO ONCORNAVIRUS-INDUCED TUMORS. (Eng.) Essex, M. (Harvard Univ. Sch. Public Health, Boston, MA 02115); Lamon, E. W. *Bibl Haematol (Basel)* (43): 166-172; 1976.

Various aspects of immunity to oncornavirus-induced tumors in several host species are reviewed. Three general classes of antigens are found on the surface of neoplastic cells from oncornavirus-induced tumors: virus structural proteins, true tumor-specific antigens, and depressed embryonic antigens. With murine leukemia virus (MuLV)-infected cells, cross-reactivity among the structural proteins may be broad or narrow, probably depending on differences in immune recognition and mechanisms of virus replication or antigen presentation. Cross-reactivity between cells infected with Friend, Moloney, and Rauscher (FMR) viruses has been demonstrated to a degree; there is relatively less reactivity with cells infected with Gross virus. Recent studies with the feline leukemia virus (FeLV) and feline sarcoma virus (FSV) indicate many characteristics in common with the murine and avian systems. With avian sarcoma virus, infected cells contain a surface glycoprotein of 100,000 daltons regardless of whether they are producing virus or not. Two levels of host interaction are considered in the immune response to virus-induced tumors: a direct response to the virus, and, failing that, a reaction to the tumor cell surface. Immune recognition of the cell surface antigens is probably partly dependent on the presence of genetic information that codes for lymphocyte receptors that can recognize antigen. The presence of virus-specific receptors may play an additional role in determining initial infectability. The thymus is also important for antigen recognition, virus infection, and/or target cell transformation. Following access to antigen, the triggering mechanism is important to determine whether potentially effective immunity or tolerance results. The effector phase of immunity to the tumor cells may involve various combinations of B and T cell mechanisms. In the murine sarcoma virus system, the cellular response in vitro involves both T and B lymphocytes. T cells are required in vivo for the regression of Moloney sarcoma virus-induced tumors. Synergy of both humoral and cell-mediated immunity may be a factor. Low humoral antibody titers to feline oncornavirus-associated cell membrane antigen in cats with naturally occurring leukemia is probably a cause of disease, rather than an effect. It is concluded that immunosurveillance to oncornavirus-transformed tumor cells may function efficiently under natural conditions. (77 references)

2434 DIFFERENT APPROACHES TO STIMULATION OF HOST DEFENSE. (Eng.) Castro, J. E. In: *Host Defense Against Cancer and Its Potentiation. Proceedings of the 5th International Symposium of The Princess Takamatsu Cancer Research Fund, Tokyo,* 1975. (Baltimore: Univ. Park Press): pp. 157-179; 1975.

Four methods of stimulating host defense to autochthonous tumors are discussed: increasing cytotoxic antibodies, suppressing serum blocking factors; increasing macrophage function, and increasing cell mediated immunity. Preferential production or physical separation of cytotoxic antibody is not always possible, but potentially cytotoxic sera can be assessed in vitro before use in vivo. Localization of cytotoxic antibodies is improved by combination with other chemicals, by selective perfusion of tumors, or by combination with other immunological manipulations. The exact nature of the serum blocking factors that stimulate tumor growth is not clear, but antibody-forming cells may be suppressed selectively by chemotherapy or alloantisera and circulating inhibitory factors, by immunoadsorption. Resistance to tumors parallels reticuloendothelial phagocytic activity, but the mechanism of these effects is not clear. There are several ways to stimulate cell mediated immunity: adoptive transfer of immunized allogeneic lymphocytes; alteration of the antigenicity of tumor cells by acetoacetylation, gluteraldehyde treatment, enzyme degradation of the cell membrane, or by incorporation of viruses into the cells; and conjugation of tumor cells with hapten. Most immunological adjuvants stimulate cellular and humoral immunity, but lentinan and orchidectomy may have selective effects upon cell-mediated immunity. It is concluded that elimination of tumors may result from alteration of several different and opposing components of the immune response; a unifying hypothesis of immune stimulation is not possible at present. (87 references)

2435 DO TUMORS GROW BECAUSE OF THE IMMUNE RESPONSE OF THE HOST? (Eng.) Prehn, R. T. (Inst. Cancer Res., Fox Chase Cancer Center, Philadelphia, PA). *Transplant Rev* 28: 34-42; 1976.

Experimental data which question the more widely held assumptions concerning the efficacy of immunological surveillance are reviewed. It appears that sporadically occurring, low incidence ''spontaneous'' tumors usually have little or no immunogenicity, which suggests that the immunogenicity of a tumor is the result of the direct action of the oncogen on the tumor cells. Several observations also argue against the possibility that immunological defenses would be effective against a very small nidus of in situ tumor: such tumors probably do not immunize the host appreciably; an extremely small tumor inoculum may grow in an experimental host, while a larger one may fail; and a very weak immune reaction may actually stimulate tumor growth (immunostimulation). There is some suggestion, however, that immunodepression is associated with a greater susceptibility to chemical oncogenesis, although not to spontaneous tumors. Allograft experiments in nude mice and in thymectomized adult mice exposed to x-irradiation suggest that while moderate thymic disability increases susceptibility to an oncogene, extreme disability depresses susceptibility to the normal range or even further. These data suggest that immunostimulation rather than surveillance may be the dominant effect of the immune response in those tumor systems in which the tumors are sufficiently immunogenic to be affected by depression of the immune reaction. Similarly, chemotherapeutic agents may be effective in part because they damage the immune reaction, which in many cases probably stimulates tumor growth. (42 references)

2436 BENIGN AND MALIGNANT PROLIFERATIVE RESPONSE: DERMAL INFILTRATION, ORIGIN AND IMPORTANCE. (Eng.) Steigleder, G. K. (Dept. Dermatology, Univ. Cologne, 5 Cologne 41, Joseph-Stelzmann-Strasse 9, W. Germany). *Acta Derm Venerol (Stockh)* 56(1): 33-41; 1976.

Lymphoreticular infiltration (LRI) is the most common type of dermal infiltration. The LRI is made up of two main components: a dermal skeleton and the infiltration itself. The dermal skeleton consists of a blood vessel, most probably a post-capillary venule, and activated fibroblasts. The activated fibroblasts contain ''paraplasmatic granules'' in their processes. Since the fibroblasts are in mutual contact by their ramifications, they are reticulum cells by definition. It is thought that they are the only local reticulum cells in the dermis. They are not stem cells, they do not belong to the reticuloendothelial system, and they do not give rise to histiocytes. Other cells, predominantly monocytes and lymphocytes, infiltrate the skeleton of the LRI system. The LRI may specialize and contain various amounts of neutrophilic, basophilic, or eosinophilic leukocytes and plasma cells. If enough B lymphocytes and plasma cells are involved, abnormal globulins may be produced (e.g., syphilis II). The lymphomas are considered to be malignant proliferative LRI-type disorders. LRI is found in allergic spreading reactions mediated by stimulated lymphocytes. A similar spreading of lymphocytes is to be expected if the allergen sensitizes a lymphocyte with neoplastic information. The lymphocyte then returns to the lymph node and gives rise to a generation of lymphocytes that spreads to the appropriate target. The degree of dedifferentiation and the response of the organism to the dedifferentiated lymphocytes are the reasons for the variety of lymphomas. (57 references)

2437 THE CONCEPT OF IMMUNOLOGICAL SURVEILLANCE AGAINST NEOPLASIA. (Eng.) Moeller, G. (Div. Immunobiology, Wallenberglaboratory, Karolinska Institutet, 10405 Stockholm 50, Sweden); Moeller, E. *Transplant Rev* 28: 3-16; 1976.

Data questioning the concept of immunological surveillance against neoplasia are reviewed. One important finding which casts doubt on the concept is that nearly all tumors and lymphoid neoplasms in man, as well as several animal tumors, are monoclonal. Of various reasons why tumors are monoclonal, the most likely is that carcinogens accelerate the appearance of rare genetic changes leading to neoplasia. This concept implies that mutagenicity and carcinogenicity should occur in parallel, and evidence strongly supports this view. Various other findings argue against the immune surveillance concept: monoclonality of tumors generally occurs in generally and locally immunosuppressed individuals; clonality occurs in tumors induced in vitro; and a logical consequence of the surveillance hypothesis (that tumors induced in vitro should possess on the average stronger tumor-specific antigens) has not been upheld by the experimental data. Although, as predicted by immunosurveillance, immunodeficient patients have an increased cancer incidence, several characteristics of these tumors are unexpected based on the surveillance hypothesis. Thus, it appears that the main mechanism of tumor appearance is a genetic mechanism analogous to somatic mutation. The T cell function appears to be to defend the individual against virus and other types of microbial infection, including tumor neoplasms; if this system fails, the individual may rarely become infected with tumor viruses which will induce neoplasms. As to the relationship between the immune system and tumors, it is unlikely that the first appearing tumor cells are recognized or rejected immunologically, hereditary neoplasms are polyclonal in patients with an intact immune system, and neoplasia is not particularly prevalent in patients with specific HL-A alleles as would be expected if the IR-gene regulated T cell function of immune surveillance plays a role in the etiology of cancer. Furthermore, escape from immunological surveillance proves nothing, since escape as well as surveillance requires the same immunization. (18 references)

2438 ROLE OF IMMUNOSURVEILLANCE AGAINST CHEMICALLY INDUCED RAT TUMORS. (Eng.) Baldwin, R. W. (Cancer Res. Campaign Lab., Univ. Nottingham, Nottingham NG7 2RD, England). *Transplant Rev* 28: 62-74; 1976.

The role of immunosurveillance against chemically-induced rat tumors is reviewed. Chemically-induced tumors in rats express diverse types of neoantigens, some of which can function to produce tumor rejection, at least as defined against transplanted tumor cells. In addition, host macrophages can control or arrest the growth of these tumors when an appropriate stimulus is provided. A variety of antagonistic processes proposed to account for the failure of immune rejection of tumor cells have been validated by in vitro methods for detecting cell-mediated immunity. There is also evidence, albeit limited, to indicate that at least some of these inhibitory processes may be operative in vivo, and these studies support the view that tumor-antigen-containing moieties may be particularly important. However, almost all of this evidence has been derived from studies on transplanted tumors in syngeneic hosts, and it remains to be shown whether effective reactions are evoked against nascent tumors developing from carcinogen-transformed cells. Even if these abrogating factors are important against transformed cells, the clear-cut differences in the responses to chemical carcinogens and oncogenic viruses in immunosuppressed hosts must be accounted for. It is evident that the immune response to neoplastic cells is multifactorial, involving the stimulation of cytotoxic or cytostatic effector cells as well as other cells suppressing these responses. This, together with the production of tumor-related products which interfere with cell-mediated immunity, indicates that the interpretation of chemical carcinogenesis in deliberately immunosuppressed hosts may not provide definitive evidence for the role of immunosurveillance in neoplasia. (47 references)

2439 THE NUDE MOUSE VS. THE HYPOTHESIS OF IMMUNOLOGICAL SURVEILLANCE. (Eng.) Rygaard, J. (Pathological Anatomical Inst., Kommunehospitalet, O. Farimagsgade 5, Copenhagen K, Denmark); Povlsen, C. O. *Transplant Rev* 28: 43-61; 1976.

The nude mouse is characterized in terms of its developmental defect, its interaction with the environment, its pathology, and selected immune responses. The defect in the nude mouse is probably due to the lack of a functional thymic epithelium. Peripheral blood, bone marrow, thoracic duct lymph, and lymphoid tissues show lymphocyte depletion, and few if any lymphocytes in the lymphoid tissues carry the membrane theta-alloantigen. Immunoglobulin M (IgM) levels are normal, but IgG and IgA levels are generally low, as are responses to thymus-dependent antigens. Nude mice readily and permanently accept foreign tissue grafts and transplanted malignant tumors. Nude mice lacking a thymus are much more susceptible to infections than are mice with normal T-cell function, but are similar to normal mice in their susceptibility to chemically-induced tumors. In contrast, they are much more sensitive than normals to oncogenic virus activity, although they show a near total absence of spontaneous malignant tumors. Nude mice will live for 4-6 mo under conventional conditions, and for over 20 mo under germfree conditions. At a time dependent on the quality of the milieu, all nude mice display the wasting syndrome and die shortly afterwards. It appears that the main pressure on nude mice is infectious disease, their life span being closely correlated with the quality of their milieu. Somatic mutation of malignancies as the constant stimulus for cell-mediated immune function (immunological surveillance) is not supported by the available data. (66 references)

2440 IMMUNOTHERAPY AND HUMAN TUMOR IMMUNOLOGY. (Eng.) Fahey, J. L. (Dept. Microbiology and Immunology, UCLA Sch. Medicine, Los Angeles, CA 90024); Brosman, S.; Ossorio, R. C.; O'Toole, C.; Zighelboim, J. *Ann Intern Med* 84(4): 454-465; 1976.

An edited transcription of a clinical case conference includes representative examples of the immune response to human tumor antigens, the effect of cancer on immune and inflammatory response capacity, and the immunotherapy and immunopharmacology of cancer in man. At least two forms of cellular immunity can be found after exposure to foreign cells: (a) the well-known cell-mediated immunity attributable to T-lymphoid cells; and (b) an antibody-dependent cellular immunity. The interaction of three components is necessary for antibody-dependent cellular immunity: target antigens, specific antibodies directed against the target antigens, and effector cells. Lymphocyte-dependent antibodies have been reported in patients suffering from several types of hematologic malignancies and in some patients with solid tumors. Cell-mediated immunity to carcinoma of the bladder has been correlated to both the stage of the disease and the type of therapy the patient received. In patients with transition cell carcinoma of the bladder, tumor-specific cell-mediated immunity was detected most frequently in patients with T1-T2 tumors. Additional approaches to immunotherapy which are under consideration include modified tumor (e.g. by neuraminidase) or tumor antigen vaccines, tissue culture lines of tumor cells for administration to patients, in vitro sensitization of lymphocytes, transfer factor and ''immune'' RNA, bone-marrow transplantation, and therapy with immune serum. (96 references)

2441 SOME IMPACTS OF CLINICAL INVESTIGATION ON IMMUNOLOGY: SURFACE IGD, IGE, AND HEAVY-CHAIN VARIANTS. (Eng.) Franklin, E. C. (New York Univ. Medical Center, 550 First Ave., New York, NY 10016). *N Engl J Med* 294(10): 531-537; 1976.

Examples of the impact of clinical observations on advances in immunology are reviewed. Studies of genetic polymorphisms have demonstrated the existence of a series of closely linked genes controlling the heavy immunoglobulin chains. The two kinds of light chains appear to be under the control of two genes, which are not linked to each other or to the heavy-chain genes, and may even be on separate chromosomes. Similar studies of genetic polymorphisms of murine and rabbit immunoglobulins have established that all immunoglobulin polypeptide chains are under the control of at least two independent genes, one of which controls the variable region and the other the constant region. The clinical features of three types of heavy chain disease (HCD), γHCD, αHCD, and μHCD, are described. γHCD occurs primarily in older people and resembles a lymphoma more than multiple myeloma. αHCD appears to be more common than γHCD and occurs most often in younger people. μHCD generally accompanies chronic lymphocytic leukemia. The diagnosis in all three cases is an immunochemical one. Structural studies of the HCD proteins have indicated that all the molecules have internal deletions of variable size and that the hinge region is where the deletion frequently ends and, on occasion, begins. Recent studies have demonstrated IgD to be a major constituent of lymphocyte membranes, probably with an important role in antigen recognition and in modulating the immune response. These studies also demonstrated the simultaneous occurrence of both IgM and IgD on the surface of a high percentage of normal and pathologic B lymphocytes. It is possible that IgD and IgM are produced at different stages of lymphocyte development. IgE, the most recent immunoglobulin to be discovered, appears to play an important role in host defense against parasitic infections. The release of various vasoactive substances resulting from the interaction of antigen with IgE bound to mast cells and basophils may alter vascular permeability, thereby facilitating the passage of other antibodies to sites where they are needed. (13 references)

2442 PRIMARY IMMUNODEFICIENCY AND MALIGNANCY. (Eng.) Kersey, J. H. (Univ. Minnesota, Minneapolis, MN); Spector, B. D.; Good, R. A. *Birth Defects* 11(1): 289-298; 1975.

Evidence indicating an essential relationship between immunological deviation and cancer is reviewed. Both humoral and cell-mediated responses are important in the control of transformed cells and pathological conditions with B lymphocyte defects, such as X-linked (Bruton) agammaglobulinemia and IgA deficiency, or with both B and T lymphocyte defects, such as Down's syndrome, common variable immunodeficiency, ataxia telangiectasia, Wiskott-Aldrich syndrome, combined system immunodeficiency, or drug-induced immunosuppression, which are associated with high rates of malignancy. The predominance of leukemias and lymphoreticular malignancies (75% of total neoplasms) in individuals with primary immunodeficiency syndromes suggests that additional factors affecting the lymphoid system may be operative in such cases, apart from failure of the immune system to suppress spontaneously generated tumors. Chronic antigenic stimulation of the lymphoid system, lack of immunoregulatory feedback mechanisms, undefined graft-versus-host reactions, or infection with (or activation of) endogenous viruses may be of some importance, and this is suggested by experimental models. (123 references)

2443 FACTORS IN THE IMMUNE RESPONSE TO TUMORS AND POTENTIAL METHODS FOR INCREASING THEIR EFFECTIVENESS. (Eng.) Amos, D. B. In: *Host Defense Against Cancer and Its Potentiation. Proceedings of the 5th International Symposium of The Princess Takamatsu Cancer Research Fund, Tokyo, 1975*. Baltimore: Univ. Park Press): pp. 143-155; 1975.

The effect of lymphocyte-mediated cytotoxicity on tumor cell cytolysis was investigated in BALB/c mice, and the influence of antibody on tumor immunity is discussed. Ascite tumors were produced in mice by transplanting lymphocytes or sarcomas in the peritoneal cavity. After several transfers, many free-floating cells were produced. When these cells were transferred to a new host, they divided exponentially for 8-10 days. In the compatible (syngeneic) host, growth continued, but at a slower rate. In the incompatible (allogeneic) host, there was an abrupt fall in the number of tumor cells, and none were found after the 11th day. Lymphocytes from the peritoneal cavity of mice that have rejected an allogeneic tumor are extremely powerful killers of tumor cells in vitro. Cytolysis or the actual killing stage is very dependent upon successful binding, a serum lipoprotein, and temperature. The peritoneal lymphocytes are capable of transferring immunity to another animal; when these lymphocytes are mixed with tumor cells, the recipient develops his own immunity and his own killer cells. The lymphocyte-antibody-lymphocyte interaction (LALI) provides an explanation for an in vivo observation in mice. Lymph node cells from C57Bl mice immune to C3H ascites sarcoma BP8 harvested 14 days after immunization have relatively little cytotoxic potential in adoptive transfer tests. These cells, when mixed with an enhancing dose (1 μl) of immune serum, produced a dramatic cytotoxic effect. This synergistic effect was not seen if the lymphoid cells were harvested 5 days after immunization. It seems likely that the antibody was being used in LALI by the injected lymphocytes. (37 references)

2444 DISORDERS OF MONONUCLEAR PHAGOCYTE PROLIFERATION, MATURATION AND FUNCTION. (Eng.) Golde, D. W. (No affiliation given). *Clin Haematol* 4(3): 705-721; 1975.

The disorders of mononuclear phagocyte proliferation, maturation, and function are analyzed on a pathophysiological basis. Proliferative disorders include reactive hyperplasia, malignant histiocytic disorders, and idiopathic histiocytoses. Reactive hyperplasia can occur in response to a variety of organisms, such as those causing tuberculosis, syphilis, brucellosis, listeriosis, leprosy, malaria, schistosomiasis, leishmaniasis, cryptococcosis, histoplasmosis, and toxoplasmosis. Histiocytic responses also occur in response to beryllium and zirconium salts, and excessive hematopoietic cell destruction may occur in acquired hemolytic anemia, congenital hemolytic syndromes, pulmonary hemosiderosis, and in diseases with a high turnover and destruction of granulocytes and platelets. A wide variety of diseases may also be associated with increased numbers of circulating monocytes. The malignant histiocytoses are a group of disorders characterized by the neoplastic proliferation of cells of the monocyte-macrophage complex; these disorders include monocytic leukemia, histiocytic lymphoma (reticulum cell sarcoma), Letterer-Siwe disease, histiocytic medullary reticulosis, and leukemic reticuloendotheliosis (hair-cell leukemia). The idiopathic histiocytoses are characterized by accumulations of well-differentiated macrophages. Wegener's granulomatosis and sarcoidosis are typical examples of this group, while Hand-Schuller-Christian disease and eosinophilic granuloma are usually classified with the histiocytoses. In generalized metabolic disorders characterized as ''storage diseases'', the catabolic capacity of the macrophage may be exceeded because of a primary defect in enzyme function or by overloading an otherwise normal cell. Storage diseases include Gaucher and Neimann-Pick diseases and sea-blue histiocytosis. Among the macrophage dysfunction syndromes are chronic granulomatous disease, myelomonocytic leukemia, and lymphoma; defective mononuclear cell function is also thought to occur in lepromatous leprosy, disseminated tuberculosis, disseminated fungal disease, and Hodgkin's disease. (94 references)

2445 WISKOTT-ALDRICH SYNDROME. (Eng.) Nevin, N. C. In: *Cancer Genetics*. Edited by H. T. Lynch. (Springfield, IL: Charles C. Thomas): pp. 249-256; 1976.

A review is presented of the clinical and pathological features, treatment, and predisposition to malignancy of patients with Wiskott-Aldrich syndrome, a severe disease of infancy and childhood. It is inherited in a sex-linked recessive manner that is characterized by eczema, thrombocytopenia, and recurrent infection. Patients have immunological disturbances of varying degrees; both humoral and cellular response systems are affected. Lymphoreticular malignancies have been reported in more than 10% of patients. The oncogenetic mechanism of the disease is not known, but the recurrent infection resulting in hyperplasia of reticulum cells may be of etiological importance. (39 references)

2446 ATAXIA-TELANGIECTASIA (LOUIS-BAR SYNDROME). (Eng.) Brown, L. R. (Dept. Diagnostic Roentgenology, Mayo Clinic and Mayo Foundation, Rochester, MN 55901); Coulam, C. M.; Reese, D. F. *Semin Roentgenol* 11(1): 67-70; 1976.

The features of ataxia-telangiectasia are reviewed. The earliest sign is often an ataxic gait in early childhood, which progressively worsens. Choreoathetosis, myoclonic jerks, dysarthria, and other neurologic abnormalities often occur. There is degeneration of Purkinje's cells and, to a lesser extent, basket and granular cells of the cerebellum. Gross atrophy of the vermis is common. Telangiectases usually appear later than the ataxia (usually at 2-8 yr of age), beginning with the eyes and progressing to the bulbar conjunctiva, the face, and to the exposed areas of the limbs. They are dilated venules branching from subcapillary plexuses. The patient is predisposed to chronic recurring sinus and pulmonary infections, otitis media, and rhinitis in many cases, due to a combined depression of multiple parts of the immune system. Roentgen findings include mucosal thickening or opacification and signs of chronic lung infections such as bronchiectasis and fibrosis. The adenoids appear small on lateral view, and the thymus is small or absent. The incidence of malignant tumors in this disease is higher than in any other primary immunodeficiency disease, approaching 10%. Most of the tumors are lymphoreticular, but about 11% are nonlymphoid. (15 references)

2447 MULTIPLE ENDOCRINE ADENOMATOSIS SYNDROMES. (Eng.) Schimke, R. N. (Univ. Kansas Hosp., Kansas City, KS). *Adv Intern Med* 21: 249-265; 1976.

The clinical characteristics, treatment, and pathogenesis of multiple endocrine adenomatosis syndromes are discussed. The association of tumors of the pituitary, pancreas, and parathyroid glands has been designated as multiple endocrine adenomatosis, type I (MEA I) or Wermer's syndrome. The adrenal glands and thyroid are less frequently affected in the MEA I syndrome, and benign and malignant bronchial and intestinal carcinoid tumors, multiple lipomas, schwannomas, and thymomas occur even more rarely. MEA I is inherited as an autosomal dominant effect with probably nearly complete penetrance of the defective gene. Considerable phenotypic variability exists even within the same family, but hormone radioimmunoassay has greatly facilitated diagnosis in asymptomatic individuals and the syndrome can often be expeditiously treated. It is suggested that all ectopic hormone production results from the faulty differentiation of neuroectoderm, either as a primary genetic effect, as in MEA I, or as a secondarily acquired phenomenon. MEA II, or the Sipple syndrome, comprises tumors of the thyroid and adrenal medulla. Occasionally, parathyroid adenomas or hyperplasia are also seen, and secondary adrenal hyperplasia has been described on rare occasions. MEA II is distinct from MEA I but, like the latter, is an autosomal dominant disorder which is a form of neuroectodermal dysplasia. Knowledge gained from these conditions may lead to the development of effective therapy for a host of hormone-producing tumors. (64 references)

2448 TUMORS OF THE ESOPHAGUS. (Fre.) Fontaine, J.-L. (5, square Raynouard, 78150 Rocquencourt, France). *Rev Med* 16(31-32): 2121-2125; 1975.

Cancers of the esophagus, 50% of which appear in the lower third, 30% in the middle, and 20% in the upper third are surveyed. Because of the extensive lymphatic system and numerous anastomoses in the esophageal area, metastasis and tumor dissemination are common. The clinical signs are dehydration, malnutrition, local pain, vomiting, halitosis, sialorrhea, and hemorrhaging. In addition to these are repetitive pertussoid attacks (indicating tracheal extension), expectoration, massive pulmonary infection, subclavicular ganglia, irregular hepatomegaly, and occasional recurrent paralysis. The tumor, which is best diagnosed by clinical examination, radiology, and endoscopy, evolves from a permanent irregularity on the esophageal wall to advanced cancer; this is followed by neoplastic infiltration. The effectiveness of treatment, surgical excision and radiotherapy, depends on the location and extent of the tumor (which may be an evolutive tumor, a benign tumor, or a sarcoma) and on the condition of the patient; treatment frequently cannot be applied. Palliative treatment for inextricable tumors are: palliative esogastric anastomoses; gastrostomy; jejunostomy; esophageal catheterization; and prolonged parenteral alimentation. Prognosis is poor. (No references)

2449 CURRENT APPROACH TO GASTRIC CANCER. (Fre.) Bernades, P. (Hopital Beaujon, Clichy, France). *Concours Med* 97(41): 6557-6568; 1975.

Literature concerning cancer of the stomach, the most prevalent digestive cancer (especially in males) but the frequency of which is declining, is reviewed. The etiological factors that best explain geographic differences in frequency are environmental and genetic. The conditions that cause predisposition to the cancer are atrophy of the gastric mucosa, Menetrier's giant hypertrophic gastritis, gastric polyadenomas, degeneration of benign gastric ulcers, and partial gastric resection for benign ulcers. Emaciation, microcytic anemia, diarrhea or constipation, vomiting, dysphagia, acanthrosis nigricans, and an occasional fever are the clinical symptoms. Radiography, with the aid of endoscopy, is valuable for diagnosis. Patients with cancer of the stomach, especially those with hepatic metastases, may have α-fetoprotein. Particular anatomical clinical forms are cancer of the cardia, cancer of the pylorus, superficial cancer, and gastric loci of malignant lymphomas. Although prognosis is generally poor, it is augmented by early diagnosis and treatment, which is usually surgical intervention. (16 references)

2450 CANCERS OF THE PANCREAS AND BILIARY TRACT: EPIDEMIOLOGICAL CONSIDERATIONS. (Eng.) Fraumeni, J. F., Jr. (Natl. Cancer Inst., Bethesda, MD 20014). *Cancer Res* 35(11/Part 2): 3437-3446; 1975.

Epidemiological patterns for pancreatic and biliary cancers reveal more differences than similarities. Pancreatic carcinoma is common in western countries, although two Polynesian groups (New Zealand Maoris and native Hawaiians) have the highest rates internationally. In the United States, the disease is rising in frequency, predominating in men and in blacks. The rates are elevated in urban areas. The origin of pancreatic cancer is obscure, but a two-fold increased risk has been documented for cigarette smokers and diabetic patients. Alcohol, occupational agents, and dietary fat have been suspected to be risk factors. The reported incidence of biliary tract cancer is highest in Latin American populations and American Indians. Gallbladder tumors predominate in women around the world. In the United States, the rates are higher in whites than blacks, and clusters of high-risk counties have been found in the north central region, the southwest, and Appalachia. The distribution of biliary tumors parallels that of cholesterol gallstones (the major risk factor for biliary cancer). Insights into biliary carcinogenesis depend upon clarification of lithogenic influences, such as pregnancy, obesity, and hyperlipoproteinemia, exogenous estrogens, familial tendencies, and ethnic-geographic factors that may reflect dietary habits. Noncalculous risk factors for biliary cancer include ulcerative colitis, clonorchiasis, Gardner's syndrome, and probably certain industrial exposures. Within the biliary tract, tumors of the gallbladder and bile duct show epidemiological distinctions. Bile duct neoplasms predominate in men; they are less often associated with stones and more often with other risk factors. In some respects, bile duct and pancreatic tumors are alike. The male predominance of both tumors, an association between cholecystectomy and pancreatic cancer, and other considerations have prompted the notion that the same biliary carcinogens may affect the bile duct, ampulla of Vater, or (by reflux) the pancreatic duct. (77 references).

2451 FORMAL DISCUSSION OF ''CANCERS OF THE PANCREAS AND BILIARY TRACT: EPIDEMIOLOGICAL CONSIDERATIONS.''

(Eng.) Goldsmith, E. I. (Cornell Medical Center, New York, NY 10021). *Cancer Res* 35(11/Part 2): 3447-3449; 1975.

The present status of carcinoma of the pancreas is dismal and becoming worse, because treatment is not improving (while incidence is increasing). Epidemiological data suggested some new ways to look at the disease. High-risk groups in the population can be identified, but it is questionable whether screening is feasible with presently available diagnostic methods. If the suggestion of a tumor-specific protein is correct, it might become reasonable to explore such screening. Although surgical removal of these tumors has become safer and more effective and total pancreatectomy will be used more extensively in coming years, surgery cannot be expected to make an important impact on the disease, since 90% of patients are inoperable when first seen. Because prodromal and/or early malignant changes are not observable in human patients, one should turn vigorously to the creation of an animal model for biliary tract and/or pancreatic tumors. Existing evidence illustrates the close similarity of baboons to humans in bile chemistry and physiology. Pancreatic cancer does occur in animals and suggests that carnivores and/or omnivores might be better candidates than herbivores to serve as animal models. Husbandry, management, and economic consideration make it entirely reasonable to turn to larger animals rather than mega-mice (if one is satisfied that phylogenetic similarity to humans and longer life-span are important). (2 references).

2452 RECTAL POLYPS AND THEIR RELATIONSHIP TO CANCER.

(Eng.) Whitehead, R. (No affiliation given). *Clin Gastroenterol* 4(3): 545-562; 1975.

The relationship between rectal polyps and cancer is discussed. Polypoid lesions may be roughly subdivided into epithelial and nonepithelial types. The nonepithelial polyps include the lymphoid, leiomyomatous, lipomatous, neurofibromatous, and other rarer types; the epithelial polyps include the the inflammatory pseudopolyps, the heterotopic polyps, the hamartomatous (juvenile and Peutz-Jegher) polyps, the metaplastic polyps, and the neoplastic (adenomatous and carcinomatous) polyps. The chances of carcinoma-in-situ behaving in an aggressive way are negligible; serial sections of the whole lesion can be used to determine whether a polypoidal lesion has invaded beyond the muscularis mucosa. All polyps should be removed in toto with a margin of healthy mucosa, and if invasion beyond the muscularis mucosa is present, a resection is required. Comparisons of the invading cells with the appearances of those in the overlying polyp usually enable the differentiation between pseudocarcinomatous invasion at the base of polyps and true carcinomatous invasion. Several lines of evidence indicate that adenomatous polyps are premalignant lesions, but it is unknown what percentage become carcinomas. There is evidence that tumors with the villous type of growth pattern have the greatest malignant potential. (15 references)

2453 WILMS' TUMOR IN DOMESTICATED AND EXPERIMENTAL ANIMALS AND IN VITRO.

(Eng.) Pirtle, E. C. In: *Wilms' Tumor*. Edited by C. Pochedly and D. Miller. (New York: John Wiley & Sons): pp. 81-101; 1976.

Literature concerning spontaneous nephroblastomas, the experimental induction of nephroblastomas, and the results obtained from in vitro investigation of Wilms' tumor and nephroblastomas, all in lower animals, is reviewed. Spontaneous nephroblastomas of embryonal origin, histologically related to Wilms' tumor in humans, occur in a wide range of mammalian species and other lower animals (swine, chicken, cattle, rats, and dogs). Nephroblastomas of animal species have a much lower potential for metastasizing when compared to Wilm's tumor in humans. Regardless of the species, all elements of the tumor arise from primitive nephrogenic blastema of mesodermal origin. These tumors can be experimentally induced in animals by transplantation of fetal renal blastema, by transplantation of tumor elements, by inoculation with viruses of the avian leukosis complex, and by feeding or inoculating a variety of chemicals. Wilms' tumor of humans and nephroblastomas of lower animals can be successfully explanted to grow in cell culture in vitro. Human and animal nephroblastomas may be essentially diploid in early passages in cell culture, but become aneuploid with continued subculturing. (88 references)

2454 THE EPIDEMIOLOGY OF CANCERS OF THE BREAST AND REPRODUCTIVE SYSTEM.

(Eng.) Doll, R. (Univ. Oxford, Oxford, England). *Scott Med J* 20(6): 305-315; 1975.

The epidemiology of cancers of the breast and of the male and female reproductive system is reviewed. Cancer of the testis consists primarily of teratoma and seminoma types, both of which occur between 20 and 50 yr of age. Testicular cancer is unknown among Blacks and is two to three times more common among Caucasians, Maoris, and Hawaiians than among Japanese, Chinese, and Indians. The differences are probably both genetic and environmental in origin. Cancer of the prostate is rare among Japanese, Chinese, and Indians and is more common among Blacks than Caucasians. The differences are probably environmental in origin, the responsible agent probably being hormonal in character. Breast cancer is more common in Western Europe and North America than in Asia and Africa, is more common in nullipara than in multiparas, and increases in incidence with progress up the socioeconomic scale. The pattern of age-specific incidence rates differs in different geographic areas. The etiology is multifactorial, although hormonal activity is certainly important. Cancer of the ovary and corpus uteri are also more common in developed countries, in nulliparas, and in women of high socioeconomic status. Cancer of the corpus tends to be associated with estrogen-secreting tumors of the ovary and, possibly, with reserpine use, while a genetic factor affecting susceptibility of cancer of the ovary is suggested. A venereal and presumably a viral cause is likely for cancer of the uterine cervix, but such a virus has never been identified. Personal cleanliness and obstructive methods of contraception appear to be aids to prophylaxis. (39 references)

2455 ULTRASTRUCTURE OF MURINE TERATOCARCINOMAS.

(Eng.) Damjanov, I.; Solter, D. In: *Teratomas and Differentiation.* Edited by M. I. Sherman and D. Solter. (New York: Academic Press, Inc.): pp. 209-220; 1975.

Electron microscopic and histochemical studies have elucidated various aspects of teratoma formation. Studies of the stem cells of testicular, ovarian, and embryo-derived teratocarcinomas have demonstrated that these cells resemble each other despite their different origins. There may thus be a common developmental pathway leading from germ cells or embryos to teratocarcinoma. The pluripotent stem line equivalent to ectodermal cells from a 7-day mouse egg cylinder would represent the common ancestral cell of all murine teratocarcinomas. In such cells the nucleus contains a large nucleolus, and euchromatin predominates over heterochromatin. The cytoplasm is replete with free ribosomes; there are few mitochondria. Embryonic cells that have passed the stage of egg-cylinder formation lose their pluripotency, become more developmentally restricted, and cannot proliferate and retain their undifferentiated character. However, in addition to rapidly proliferating stem cells, teratomas do contain various somatic tissues derived from stem cells. Such somatic tissues show remarkable variation in structural organization, maturity, and homogeneity. Tissues formed through complex morphogenetic interactions (e.g., lung and kidney) are never found. As in malignant brain tumors, the otherwise ubiquitous enzyme, alkaline phosphatase, could not be detected in the endothelial cells of blood vessels within teratocarcinomas. (33 references)

2456 CONTROL OF TERATOCARCINOGENESIS.

(Eng.) Solter, D.; Adams, N.; Damjanov, I.; Koprowski, H. In: *Teratomas and Differentiation.* Edited by M. I. Sherman and D. Solter. (New York: Academic Press, Inc.): pp. 139-159; 1975.

The influence of both genetic and environmental factors on the development of spontaneous and experimentally induced teratomas and malignant teratocarcinomas is discussed. Genetic factors play an important role in spontaneous teratocarcinogenesis in mice: about 1% of all male mice of strain 129 develop testicular teratomas, and about 50% of female mice of the LT strain develop ovarian teratomas. However, other nongenetic factors also influence the development of these teratomas, which, for instance, occur with different frequencies in the left and right gonads and with greater frequencies in second or later litters. Experimental teratomas can be produced in mice by grafting the genital ridges isolated from embryos into adult testes or transplanting whole embryos into the testis or under the kidney capsule of an isogenic host. Genetic factors and the age and site of the graft influence the incidence of the grafted genital ridges that develop into teratomas. Grafted embryos, if they grow at all, always develop into teratomas. The host exerts genetic influence on their development into teratocarcinomas. The mechanism by which this genetic influence is expressed is unknown, but it is suggested that the reaction of the host immune system to the presence of undifferentiated cells, and the effect of such a reaction on the subsequent development of these cells, may be involved. (33 references)

2457 HODGKIN'S DISEASE: A CURABLE CANCER.

(Fre.) Rozencweig, M. (Universite Libre de Bruxelles, Faculte de Medecine, Institut Jules Bordet); Kenis, Y.; Goffin, J. C.; Mendes da Costa, P. *Brux Med* 55(9): 473-481; 1975.

The prognosis of Hodgkin's disease is reviewed according to several criteria: (a) extent of tissue involvement and presence of generalized symptoms; (b) type of and infiltration of abnormal cells; (c) serum iron and copper and serum sedimentation rate; (d) mediastinum involvement; (e) age and sex of patient. Modes of treatment including local or whole body irradiation, combination chemotherapy, and splenectomy are discussed in relationship to the stages of the disease. All patients at the Jules Bordet Institute are subjected to splenectomy, exploratory laparotomy, and high-dose irradiation of chest and abdomen. Chemotherapy is instituted if further irradiation would be toxic or unbeneficial. A table is included that summarizes the use of multiple chemotherapeutic agents. Chemotherapy and radiotherapy of Hodgkin's disease have progressed to the degree that a complete cure may be achieved. (57 references)

2458 ETIOLOGY OF THE ASSOCIATION OF LEUKEMIA AND SARCOMA. (Fre.) Boyer, J. (Institut d'Hygiene et de Medecine Preventive, 15, rue de l'Ecole de Medecine, 75006 Paris, France). *Sem Hop Paris* 50(46): 2887-2893; 1974.

The concept that acute leukemia in children and that Burkitt's lymphoma are only rare complications of inevitable, immunizing viral infections that are rarely perceived is extended to include acute leukemia in adults, sarcomas, and other cancerous infections. In spite of the fact that viruses cannot be found in many human tumors, the idea of viral etiology should not be abandoned, because the virus has been found in animal tumors. Chemicals such as benzopyrene are carcinogenic in that they provoke and stimulate an already present but latent infection. The lack of contagiousness of cancer is due to the fact that these immunizing viral infections generally pass unperceived and only rarely become cancers through complication. More cancer research should be oriented in this direction. (2 references)

2459 THE GENETICS OF RETINOBLASTOMA. (Eng.) Falls, H. F. In: *Cancer Genetics*. Edited by H. T. Lynch. (Springfield, IL: Charles C. Thomas): pp. 257-262; 1976.

Retinoblastoma is a highly malignant intraocular tumor of early childhood, with approximately 8% of all cases being of hereditary origin. Transmission of the hereditary disease is autosomal dominant, with a penetrance of 20%-80%. Overall, about 65%-80% of cases are unilateral and 20%-35% bilateral, many of the bilateral cases being hereditary. Details of diagnosis, differential diagnosis with other pathological eye conditions (pseudoglioma, larval granulomatosis, retrolental fibroplasia, persistent hyperplastic primary vitreous, Coat's disease, and angiomatosis retinae), and suggestions for genetic counseling are given. (8 references)

2460 MECHANISMS OF OSTEOLYSIS AND HYPERCALCEMIA IN CANCER. (Fre.) Lemaire, V. (Hopital Lariboisiere, U.E.R. Lariboisiere - Saint-Louis, Paris VII, 2, rue A,-Pare, F 75475 Paris Cedex 10, France); Ryckewaert, A. *Nouv Presse Med* 4(45): 3167-3168; 1975.

The secretion of substances that stimulate the osteolytic processes associated with many cancers is reviewed. The cancer cells of an osteolytic metastasis are not themselves osteophagic; they either secrete or induce secretion of parathormone. Many other substances that stimulate osteoclastic resorption have been found in cancers with hypercalcemia but without osseus metastases. Certain of these extraosseus cancers with hypercalcemia accompanied by hypophosphoremia secrete, not parathormone, but its precursor, proparathormone; enzymatic cleavage of this slightly longer polypeptide yields parathormone. Certain intraosseus metastases may also secrete proparathormone. This hypercalcemia of cancers without osseus metastases is not always the result of parathormone or its precursor. As seen by experiments with mice, prostaglandin E_2 may also be a cause. Other possibilities are osteoclastic activating factor and a sterol analog of vitamin D. (19 references)

2461 THE ROLE OF CHROMOSOMAL STUDIES IN CANCER EPIDEMIOLOGY. (Eng.) Sandberg, A. A.; Sakurai, M. In: *Cancer Genetics*. Edited by H. T. Lynch. (Springfield, IL: Charles C. Thomas): pp. 122-145; 1976.

The authors discuss the relationship of visibly recognizable chromosomal changes to cancer incidence among subjects with autosomal or sex chromosome anomalies, chromosomal breakage syndromes, or acquired karyotypic changes induced by irradiation, chemical agents, or viruses. The Philadelphia chromosome (Ph'), apparently an abbreviated G22 autosome, is the best characterized example of close association between a chromosomal aberration and neoplastic disease--in this instance, chronic myelocytic leukemia (CML). Studies with twins have demonstrated that it is an acquired cytogenetic abnormality. The exact role of Ph' in the genesis of CML is not known, but it may be that whatever causes Ph' also causes CML, either independently or through the intervention of the abnormal chromosome. Chromosomal changes in frank neoplastic disease and in preneoplastic conditions are also discussed. In neoplasia, karyotypic changes are confined solely to the cancerous cells and, generally, once a cytogenetic picture is established in a neoplasia, it remains stable throughout the course of the disease. Consideration is also given to chromosomal changes brought about by x-rays and viral transformation, and to possible future approaches to chromosomal studies in human cancer epidemiology. (96 references)

2462 FUNDAMENTALS OF HUMAN GENETICS. (Eng.) Kaplan, A. R. In: *Cancer Genetics*. Edited by H. T. Lynch. (Springfield, IL: Charles C. Thomas): pp. 32-51; 1976.

A general introduction to human genetics is presented. Discussion is made of: the interaction of genetic and nongenetic factors in the etiology of pathological disorders; concepts of phenotype and genotype; pedigree analysis; penetrance, expressivity, and heritability; statistics in genetics; the Hardy-Weinberg principle; selection, drift, and mutation; linkage; multifactorial inheritance; the nature of the genetic material; cytogenetics; sex chromatin and the sex chromosomes; autosomal abnormalities; and chromosomal abnormalities, associations, and clusterings. (57 references)

2463 CHALONES AND CANCER. (Eng.) Bullough, W. S. In: *Host Defense Against Cancer and Its Potentiation. Proceedings of the 5th International Symposium of the Princess Takamatsu Cancer Research Fund, Tokyo, 1975*. (Baltimore: Univ. Park Press): pp. 317-335; 1975.

The normal mitotic control mechanism of adult mammalian tissues and its relationship to the nature of cancer are discussed. Mitotic control depends basically on the conflicting actions of a tissue-specific mitotic inhibitor that permeates the tissue (the chalone) and a non-tissue-specific mitotic promotor that acts only in cells in contact with connective tissue (the mesenchymal factor). Thus basal cells tend to be mitotic, while distal cells are forced by the high ambient chalone concentration to become postmitotic and to age and die. The basal cells are displaced distally by mitotic pressure and thus a perfect balance is maintained between cell gain and cell loss. This mechanism apparently breaks to allow tumor growth when the chalone concentration falls so low that the distal cells also tend to remain mitotic; this is caused by an abnormality of the cell membrane. Tumor cells continue to respond to the chalone of their original tissue, so that when the chalone concentration rises, the tumor stops growing. With continued chalone treatment a tumor may disappear permanently, because with the mitotic potential reduced, destructive influences such as the immune response predominate. (86 references)

2464 THE PURIFICATION AND CHEMISTRY OF PITUITARY GLYCOPROTEIN (Eng.) Liu, W.-K. (Univ. Texas System Cancer Center, M.D. Anderson Hosp. and Tumor Inst., Houston, TX 77025); Ward, D. N. *Pharmacol Ther Part B* 1(3): 545-570; 1975.

A brief review is presented of the isolation, purification, analysis, physicochemical properties, and primary amino acid sequences of the pituitary glycoprotein hormones thyrotropin (TSH), luteinizing hormone (LH), and follicle-stimulating hormone (FSH). Reported molecular weights are approximately 26,000 for TSH, 28,000-30,000 for LH, and 35,000 for FSH, each being comprised of two nonidentical subunits of similar molecular weights. The primary amino acid sequence of the α-subunit of bovine TSH was found to be the same as that of the α-subunits of bovine and ovine LH, with two carbohydrate moieties attached to the asparagine residues 56 and 82. The β-subunit of bovine TSH differs from that of LH. The α-subunit of ovine FSH has a much smaller content of carbohydrate than the β-subunit, which is not the case with LH and TSH. (190 references)

2465 PSYCHOLOGICAL FACTORS IN THE ETIOLOGY AND TREATMENT OF CANCER OF THE CERVIX. (Eng.) Labrum, A. H. (Univ. Rochester Sch. Medicine and Dentistry, Rochester, NY). *Clin Obstet Gynecol* 19(2): 419-430; 1976.

Squamous cell cancer of the cervix has been limited to women who are or have been sexually active. The two most important etiological factors in determining a predisposition for cervical cancer are age at first intercourse and the number of sexual partners. Many psychological studies of such patients have demonstrated a feeling of hopelessness in these women, defined as a feeling of despair or lack of purpose in life. Childhood and adolescent experiences in such patients have corresponded with indices of life-styles ''conducive to cancer'', thus possibly leading to earlier and more frequent sexual intercourse as a pretext for being loved or needed. The woman with a more normal childhood and adolescence is only at risk for cervical cancer if she loses her partner, child, or purpose for living; however, she may have a better prognosis than the ''hopeless'' group. A discussion of the management of the terminal cancer patient is included. (32 references)

2466 STEROID HORMONE RECEPTORS: A SURVEY (Eng.) Mainwaring, W. I. P. (Imperial Cancer Res. Fund, Lincoln's Inn Fields, London, England). *Vitam Horm* 33: 223-245; 1975.

A simplified model for the action of steroid hormones, including uptake, metabolism, binding to a high-affinity receptor, activation, and molecular action, is presented and discussed. Antagonists for many steroids are now available that counter the formation of the receptor complex and negate virtually all of the hormonal response. Differences are cited in the receptor contents of experimental tumors that may exist in glucocorticoid-sensitive and -insensitive lines. Credence in receptor systems is drawn from the striking correlation between steroid-binding receptors and biological activity. Evidence for and against an entry mechanism for steroids is discussed. Steroid antagonists are of interest as potential chemotherapeutic agents and as research tools. Regarding steroid metabolism in the model system, only for the androgens can a convincing case be made for mandatory involvement. At this point, the purification of steroid-receptor complexes is urgent in further elucidation of the mechanisms of action of steroid hormones. Conventional approaches to this problem and affinity chromatography are discussed. Further effort is also required to provide a satisfactory explanation for tissue specificity of hormonal responses. (109 references)

2467 CYCLIC AMP AND CELL BEHAVIOR IN CULTURED CELLS. (Eng.) Willingham, M. C. (Lab. Molecular Biology, Natl. Cancer Inst., Bethesda, MD 20014). *Int Rev Cytol* 44: 319-363; 1976.

A comprehensive review is presented of the modulation by cAMP of the morphology, contact inhibition, membrane transport of nutrients, motility, adhesiveness, migration, and agglutination by plant lectins in cultured normal and transformed cells. Although in mature nondividing differentiated cells in vivo cAMP regulates the expression of specific differentiated functions, in growing cultured cells cAMP regulates cell growth. High levels of cAMP slow down or stop replication in both normal and transformed cultured cells, transformed cells usually possessing lower endogenous cAMP levels than their parent cell lines. In general, cAMP also reverses the effect of transformation on other cell properties, returning them toward normal. It is proposed that the alteration of cell shape and motility by cAMP is brought about by effects on microtubules and microfilamentous structures and that alterations of agglutinability are brought about by regulation of the presence of surface microvilli. (29 references)

2468 ABNORMAL REGULATION OF CYCLIC AMP BINDING PROTEINS. A CRITICAL LESION OF MALIGNANCY OF NON-NEURAL CELLS. (Eng.) Prasad, K. N. (Univ. Colorado Medical Center, 4200 E. 9th Ave., Denver, CO 80220); Sinha, P. K. *Differentiation* 6(1): 59-60; 1976.

On the basis of several reports, it is proposed that a defect in the regulation of cyclic AMP (cAMP) binding proteins may be one of the critical lesions in the cancer cells derived from glial cells and fibroblasts. Mutation in the regulatory gene for cAMP binding proteins in nonneural cells may occur as a result of viruses, chemical carcinogens, ionizing radiation, or any combination of these agents. (16 references)

2469 ESTROGEN IMPLANTATIONS APPARENTLY FOLLOWED BY PRIMARY CANCER OF THE LIVER (Letter to Editor). (Fre.) Caroli, J. (Hopital Franco-musulman, F 93009 Bobigny, France); Paraf, A. *Nouv Presse Med* 5(15): 1003; 1976. (3 references)

2470 RAUWOLFIA ALKALOIDS AND BREAST CANCER (LITERATURE REVIEW). (Dut.) Soeterboek, A. M. (No affiliation given). *Pharm Weekbl* 110(45): 1126-1128; 1975. (20 references)

2471 SQUAMOUS CELL NEOPLASTIC CHANGE IN PATIENTS WITH VAGINAL ADENOSIS (Letter to Editor). (Eng.) Burke, L. (Beth Israel Hosp., 330 Brookline Ave., Boston, MA 02215); Antonioli, D. *Am J Obstet Gynecol* 124(6): 665; 1976. (2 references)

2472 CURRENT DANGERS AND PROBLEMS IN THE TOPICAL USE OF STEROIDS. (Eng.) Deakin, M. J. (Wickham House, 155 Wickham Terrace, Brisbane, Q. 4000, Australia). *Med J Aust* 1(5): 120-121; 1976. (27 references)

2473 COSMETIC SCIENCE: 1975 LITERATURE SURVEY. (Eng.) Rieger, M. M. (Warner-Lambert Res. Inst., Morris Plains, NJ). *Cosmetics and Toiletries* 91(4): 25-36; 1976. (152 references)

2474 CAUSATIVE AGENTS IN THE INDUCTION OF BLADDER CANCER. (Eng.) Jackson, C. D. (Natl. Cancer Toxicological Res., Jefferson, AR 72079); Baetcke, K. P. *Ann Clin Lab Sci* 6(3): 223-232; 1976. (54 references)

2475 FACT AND FANCY ABOUT DRINKING WATER (Editorial). (Eng.) Stewart, W. H. (Louisiana Health and Human Resources Admin., LA). *Am J Public Health* 65(10): 1111; 1975. (1 reference)

2476 VINYL CHLORIDE: INTRODUCTION TO THE PROBLEM. (Eng.) Gauvain, S. (Medical Advisory Service, Baynards House, 1 Chepstow Place, Westbourne Grove, London W2 4TF, England). *Proc R Soc Med* 69(4): 275-277; 1976. (12 references)

2477 BIOLOGICAL EFFECTS OF N-METHYL-N'-NITRO-N-NITROSOGUANIDINE. (Eng.) Sinha, U. (Dept. Botany, Univ. Delhi, Delhi 110007, India); Chattoo, B. B. *J Sci Ind Res* 34(9): 499-505; 1976. (110 references)

2478 REPORT OF DISCUSSION GROUP NO. 13. TISSUE CULTURE TECHNIQUES. (Eng.) Weinstein, B. In: *Carcinogenesis Testing of Chemicals. Proceedings, Conference on Carcinogenesis Testing in the Development of New Drugs, May 23-25, 1973, Washington, D.C.* CRC Press, Inc. (Cleveland, OH): pp. 109-111; 1974. (17 references)

2479 ALPHA-EMITTERS (Letter to Editor). (Eng.) Elkeles, A. (London, England). *Am Sci* 63(6): 618-619; 1975. (6 references)

2480 ALPHA-EMITTERS (Letter to Editor). (Eng.) Martell, E. A. (No affiliation given). *Am Sci* 63(6): 619-620; 1975. (2 references)

2481 ONCORNA VIRUSES I: VIRUS-CELL INTERACTIONS AND VIRUS STRUCTURE ANALYSES. MAMMALIAN C-TYPE ONCORNA VIRUSES. RELATIONSHIPS BETWEEN STRUCTURAL VIRUS AND CELL SURFACE ANTIGENS AND THEIR POSSIBLE SIGNIFICANCE IN IMMUNOLOGICAL DEFENSE MECHANISMS. (Eng.) Schaefer, W. (Max-Planck-Institut fuer Virusforschung, Spemannstrasse 35/111, D-7400 Tuebingen, W. Germany); Claviez, M.; Frank, H.; Hunsmann, G.; Moennig, V.; Schwarz, H.; Thiel, H. J.; Bolognesi, D. P.; Green, R. W.; Langlois, A. J.; Fischinger, P. J.; De Noronha, F. *Bibl Haematol (Basel)* (43): 88-96; 1976. (21 references)

2482 HUMAN ACUTE LEUKEMIA: INCREASING EVIDENCE FOR INVOLVEMENT OF TYPE-C RNA TUMOR VIRUSES. (Eng.) Gallo, R. C.; Gallagher, R. E.; Mondal, H.; Gillespie, D. H. In: *Tumor Virus-Host Cell Interaction.* Edited by A. Kolber. (New York: Plenum Press): pp. 337-352; 1975. (36 references)

2483 GENERAL ASPECTS OF CELL TRANSFORMATION AND TUMOR INDUCTION BY RNA ONCOGENIC VIRUSES. (Eng.) Svec, J. (Cancer Res. Inst., Bratislava, Czechoslovakia). *Int Union Cancer Tech Rep Ser* 20: 94-97; 1975. (4 references)

2484 THE ASSOCIATION OF HERPES SIMPLEX VIRUS AND CERVICAL CANCER: A REVIEW. (Eng.) Simon, J. W. (Mount Sinai Sch. Medicine, New York, NY 10029). *Gynecol Oncol* 4(1): 108-116; 1976. (40 references)

2485 EXPRESSION OF VIRAL INFORMATION--A REVIEW. (Eng.) Hampar, B. In: *Oncogenesis and Herpesviruses II. Part 1: Biochemistry of Viral Replication and In Vitro Transformation. Proceedings of a Symposium held in Nuremburg, Federal Republic of Germany, 14-16 October 1974.* International Agency for Res. on Cancer. (Lyon, France): pp. 449-455; 1975. (43 references)

2486 THE ''SARC'' GENE. (Fre.) Manteuil-Brutlag, S. (No affiliation given). *Biochimie* 57(10): 1113-1116; 1975. (14 references)

2487 FIBROBLAST SURFACE ANTIGEN (SF): MOLECULAR PROPERTIES, DISTRIBUTION IN VITRO AND IN VIVO, AND ALTERED EXPRESSION IN TRANSFORMED CELLS. (Eng.) Vaheri, A. (Dept. Virology, Univ. Helsinki, SF-00290 Helsinki 29, Finland); Ruoslahti, E.; Linder, E.; Wartiovaara, J.; Keski-Oja, J.; Kuusela, P.; Saksela, O. *J Supramol Struct* 4(1): 63-70; 1976. (51 references)

2488 MASKING OF CELL-SURFACE ANTIGENS ON CANCER CELLS. (Eng.) Codington, J. F. In: *Cellular Membranes and Tumor Cell Behavior. A Collection of Papers Presented at the Twenty-Eighth Annual Symposium on Fundamental Cancer Research, 1975.* University of Texas System Cancer Center, M. D. Anderson Hospital and Tumor Institute. (Baltimore: Williams and Wilkins Co.): pp. 399-419; 1975. (50 references)

2489 THE INTERACTION OF HOMOGENEOUS, MURINE MYELOMA IMMUNOGLOBULINS WITH POLYSACCHARIDE ANTIGENS. (Eng.) Glaudemans, C. P. J. (Natl. Inst. Arthritis, Metabolism, Digestive Diseases, Natl. Insts. Health, Bethesda, MD 20014). *Adv Carbohydr Chem Biochem* 31: 313-346; 1975. (186 references)

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2491 MACROPHAGES, RECOGNITION FACTORS, AND NEOPLASIA. (Eng.) Di Luzio, N. R. In: *The Reticuloendothelial System.* International Acad. of Pathology Monograph. (Baltimore: Williams and Wilkins Co.): pp. 49-64; 1975. (68 references)

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2497 CHANGING PATTERNS OF CANCER. (Eng.) Anonymous. (No affiliation given). *Br Med J* 1(6005): 301-302; 1976. (7 references)

2498 PATHOLOGY OF SOLID TUMOURS IN MICE. (Eng.) Dux, A. (Netherlands Cancer Inst., Amsterdam, The Netherlands). *Int Union Cancer Tech Rep Ser* 20: 36-39; 1975. (6 references)

2499 IMMUNOLOGICAL AND ETIOLOGICAL ASPECTS OF HODGKIN'S DISEASE. (Eng.) Hirshaut, Y.; Dosik, M. H. In: *Hodgkin's Disease.* Edited by M. J. Lacher. (New York: John Wiley & Sons): pp. 387-403; 1976. (101 references)

2500 THE SIGNIFICANCE OF EXPERIMENTAL GLIOMAS FOR HUMAN DISEASE. (Eng.) Zimmerman, H. M. (No affiliation given). *Recent Results Cancer Res* 51: 6-19; 1975. (27 references)

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2502 INCIDENCE OF ENDOMETRIAL CARCINOMA (Letter to Editor). (Eng.) Doll, R. (Radcliffe Infirmary, Oxford, England); Kinlen, L. J.; Skegg, D. C. G. *Br Med J* 1(6017): 1071-1072; 1976. (4 references)

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2504 HETEROCHROMIA AND NEUROBLASTOMA (Letter to Editor). (Eng.) Jaffe, N. (Sidney Farber Cancer Center, 35 Binney St., Boston, MA 02115). *J Pediatr* 88(1): 168; 1976. (No references)

2505 HETEROCHROMIA AND NEUROBLASTOMA (Letter to Editor). (Eng.) Meadows, A. T. (Children's Hosp. Philadelphia, Philadelphia, PA 19104); Knudson, A. G., Jr. *J Pediatr* 88(1): 168; 1976. (5 references)

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2507 MALIGNANT MELANOMA OF THE FEMALE GENITALIA: A CLINICAL ANALYSIS. (Eng.) Morrow, C. P. (Women's Hosp., 1240 N. Mission Rd., Los Angeles, CA 90033); DiSaia, P. J. *Obstet Gynecol Surv* 31(4): 233-271; 1976. (187 references)

2508 MORTALITY FROM LUNG CANCER (Letter to Editor). (Eng.) Kennedy, A. (Univ. Sheffield Medical Sch., Sheffield S10 2RX, England). *Lancet* 1(7966): 965; 1976. (3 references)

2509 MORTALITY FROM LUNG CANCER (Letter to Editor). (Eng.) Myddelton, G. (Blue Moon, 1861 Glutieres-sur-Ollon, Vaud, Switzerland). *Lancet* 1(7962): 740-741; 1976. (2 references)

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2511 THE RELATION OF PYSCHOLOGICAL STRESS TO ONSET OF MEDICAL ILLNESS. (Eng.) Hurst, M. W. (Boston Univ. Medical Sch., Boston, MA 02118); Jenkins, C. D.; Rose, R. M. *Annu Rev Med* 27: 301-312; 1976. (54 references)

2512 MISQUOTATION OF ARTICLE (Letter to Editor). (Eng.) Fidler, I. J. (Frederick Cancer Res. Center, P.O. Box B, Frederick, MD 21701). *J Natl Cancer Inst* 56(2): 212-213; 1976. (3 references)

2513 CLOSING REMARKS--MEMBRANE ABERRATIONS IN NEOPLASIA: RELEVANCE TO THE MALIGNANT PROCESS. (Eng.) Wallach, D. F. H. In: *Cellular Membranes and Tumor Cell Behavior. A Collection of Papers Presented at the Twenty-Eighth Annual Symposium on Fundamental Cancer Research, 1975.* University of Texas System Cancer Center, M. D. Anderson Hospital and Tumor Institute. (Baltimore: Williams and Wilkins Co.): pp. 549-567; 1975. (32 references)

2514 THE EFFECTS OF PROTEASE INHIBITORS ON CELLS IN VITRO. (Eng.) Schnebli, H. P. In: *Proteases and Biological Control. Cold Spring Harbor Conferences on Cell Proliferation.* Cold Spring Harbor Lab. (Cold Spring Harbor, NY): vol. 2, pp. 785-794; 1975. (52 references)

2515 TRANSFER RNA METHYLASE ACTIVITY AND CAPACITY DURING AFLATOXIN B$_1$-INDUCED HEPATOCELLULAR CARCINOGENESIS.

(Eng.) Busby, W. F., Jr. (Dept. Nutrition and Food Sci., Massachusetts Inst. Technology, Cambridge, MA 02139); Paglialunga, S.; Newberne, P. M.; Wogan, G. N. *Cancer Res* 36(6): 2013-2018; 1976.

The authors determined the relationship between transfer RNA (tRNA) methylase activity and capacity and the sequential histopathological stages during hepatocellular carcinoma development in rat liver by aflatoxin B$_1$ under conditions that minimized toxic effects. The enzyme activity and capacity (indicators of the transformation and progression of preneoplastic lesions to the fully malignant state) were monitored in whole-rat-liver preparations during the induction of hepatocellular carcinomas by an 8-wk aflatoxin B$_1$ dose regimen that produced minimal toxic effects. Significant phases of elevated tRNA methylase capacity occurred at 6 to 9 wk (29%) and 24 to 29 wk (40%). No significant change in tRNA methylase activity was noted over the course of the 55-wk experiment. Higher aflatoxin B$_1$ doses, producing acute liver toxicity, resulted in elevated tRNA methylase activity (50%) and capacity (30%) at least as early as 1 wk after dosing. Experiments with individual nodular lesions excised from livers of rats continuously fed a diet containing 2 ppm aflatoxin B$_1$ demonstrated similarly elevated tRNA methylase activities and capacities in hyperplastic (preneoplastic) nodules, with and without histological evidence of carcinoma.

2516 AFLATOXIN-INDUCED TUMORS IN MICE.

(Eng.) Louria, D. B. (New Jersey Medical Sch., Newark, NJ); Finkel, G.; Smith, J. K.; Buse, M. *Sabouraudia* 12(3): 371-375; 1974.

Mice were given a mixture of aflatoxins B and G subcutaneously (100 μg/day), by force feeding (100 μg/day) or by aerosol (1 mg/ml) for up to 1 yr. Those given the aflatoxins subcutaneously developed local sarcomas in dose-related fashion. Those given aflatoxins by any route showed an incidence of lymphatic leukemia significantly greater than that of age-matched control mice. The increase in leukemia incidence was unexpected. Presumably, leukemia is virus-induced; the aflatoxin then acts either on the virus or the host. It apparently promotes the hematologic malignancy by interfering with the transcription of nuclear DNA by RNA polymerases and with replication of DNA by DNA polymerases.

2517 ASSESSMENT OF EXPOSURE TO AFLATOXINS.

(Eng.) Wogan, G. N. In: *Host Environment Interactions in the Etiology of Cancer in Man. Proceedings of a Meeting Held at Primosten, Yugoslavia, 27 August-2 September 1972.* Edited by R. Doll, I. Vodopija and W. Davis. International Agency for Research on Cancer. (Lyon, France): pp. 237-241; 1973.

The results of investigations carried out by four groups in different countries of Africa and Asia to obtain estimates of aflatoxin intake by populations in which primary carcinoma of the liver occurs at different incidences are summarized. Market samples of peanuts collected in various localities within Swaziland were analyzed for aflatoxins and aflatoxin intake estimated by interviews concerning habitual patterns of peanut ingestion. The geographical distribution of liver cancer in Swaziland derived from cancer registry data suggested a higher disease risk in those areas with highest frequency of aflatoxin contamination. In Uganda, an attempt was made to collect samples of all major diet ingredients from home granaries. When the tribal distribution of hepatoma incidence, as determined by identification of new cases, was compared to frequency of aflatoxin contamination of foodstuffs, the highest incidence of the disease was seen in tribal regions in which frequency of contamination was also high. In Kenya, aliquots of total diet and of beers (as consumed) were systematically collected and analyzed for aflatoxin. Average aflatoxin intakes were calculated on the basis of assumed daily intakes of food and beverages and, as in the other studies, the groups with higher average aflatoxin intakes also had high incidences of hepatoma. A similar study was undertaken in Thailand. In the first phase, a survey of market samples of food indicated that 9% of the sampled food was contaminated with aflatoxin. Based on the results of the market survey, three areas in Thailand representing suspected high, intermediate, and low levels of contamination were chosen for a dietary survey. A portion of the diet of selected families was collected and assayed for aflatoxins. Based on weights of cooked foods eaten, aflatoxin intake was calculated on a family basis for each region. There was a threefold greater incidence in liver cancer in the intermediate, compared to the low-aflatoxin-intake, region. This approach has significant problems and sources of error that limit its value in making quantitative estimates of population exposure. An approach obviating many of these problems would be the development of a method to determine urinary excretion of the carcinogen or its metabolite(s).

2518 MUTAGENIC ACTIVITY OF STYRENE OXIDE (1,2-EPOXYETHYLBENZENE), A PRESUMED STYRENE METABOLITE.

(Eng.) Milvy, P. (Mount Sinai Sch. Medicine, Fifth Ave. and 100th St., New York, NY 10029); Garro, A. J. *Mutat Res* 40(1): 15-18; 1976.

The possible mutagenic effects of styrene and some of its metabolites were studied in various strains of *Salmonella typhimurium*. Only styrene oxide induced mutations in strains TA1535 and TA100, showing an activity similar to vinyl chloride and its presumed metabolites. The mutants were not clustered around the zone of inhibited growth, but randomly distributed, suggesting that the styrene oxide was dispersed by vaporization rather than by diffusion through the agar. It is concluded that styrene vapor may metabolize to a potentially carcinogenic compound, and current acceptable levels may pose a health threat to workers.

2519 REACTION OF NITROSAMINES IN THE UDENFRIEND SYSTEM: PRINCIPAL PRODUCTS AND BIOLOGICAL ACTIVITY. (Eng.) Hsieh, S.-T. (Dept. Food Science Nutrition, Massachusetts Inst. Technology, Cambridge, MA 02139); Kraft, P. L.; Archer, M. C.; Tannenbaum, S. R. *Mutat Res* 35(1): 23-28; 1976.

Products formed after incubation of diethylnitrosamine (DEN) and nitrosopiperidine (NP) in the Udenfriend system (a nonenzymatic hydroxylation system consisting of ascorbic acid, ferrous ions, EDTA, and molecular oxygen) were isolated, characterized and tested for mutagenic activity with *Salmonella typhimurium* TA 1535. After incubation, the reaction mixture was extracted with dichloromethane and concentrated. The reaction products were analyzed by high-pressure liquid chromatography in a column of Corasil II with dichloromethane as eluant, or on silica gel plates. A rat liver microsomal activation system was prepared from male Sprague-Dawley rats, whose drinking water had been supplemented with 0.1% sodium phenobarbital for 1 wk prior to sacrifice. The results of thin-layer and high pressure liquid chromatography indicated that the reaction of DEN in the Udenfriend system yielded N-nitroso-2-(ethylamino)-ethanol as the major product. The major product derived from NP in the same system was N-nitroso-4-piperidone. These products were mutagenic to *S. typhimurium* TA 1535 only when activated by rat liver microsomes. Both N-nitroso-2-(ethylamino)-ethanol and N-nitroso-4-piperidone have been shown to be carcinogenic in the rat. In view of these results on mutagenicity, neither of the oxidized nitrosamines is likely to be an ultimate carcinogenic species.

2520 EXPERIMENTAL INVESTIGATIONS ON THE INFLUENCE UPON THE CHEMICAL CARCINOGENESIS. SECOND COMMUNICATION: STUDIES WITH 3,4-BENZOPYRENE. (Eng.) Schmaehl, D. (Institut fuer Toxikologie und Chemotherapie am Deutschen Krebsforschungszentrum, D-6900 Heidelberg, Im Neuenheimer Feld 280, W. Germany); Habs, M.; Diehl, B. *Z Krebsforsch* 86(1): 85-88; 1976.

To determine the effects of immunostimulants, immunodepressive agents, or neurotropic poisons on tumors induced by 3,4-benzopyrene (BP), 486 Sprague-Dawley rats (2 days old) received sc injections of 0.5 mg of BP. After 250 ± 70 days, 50% of the animals developed local fibrosarcomas. Subsequent treatment with immunostimulating vaccine (bacille Calmette Guerin, albumin, vitamin A acid) or immunodepressive agents (hydrocortisone, cyclophosphamide, methotrexat), which was started 6 days after birth and maintained throughout life, did not influence carcinogenesis with respect to tumor incidences or induction periods.

2521 EMBRYONIC LUNG EPITHELIAL-MESENCHYMAL INTERACTIONS DURING THE PROCESS OF CARCINOGENESIS. I. EFFECT OF CARCINOGENIC HYDROCARBONS. (Eng.) Bal, E. (Departamento de Investigaciones, Instituto de Oncologia "A. H. Roffo", Avda. San Martin 5481, Buenos Aires, Brazil); De Lustig, E. S. *Medicina (B Aires)* 34(1): 29-38; 1974.

The epithelial-mesenchymal interactions between lung tissues following treatment with 3,4-benzo(a)-pyrene (BP) or 9,10-dimethylbenzanthracene (DMBA) were studied in vitro. Six-day-old chick lung epithelium was mechanically dissociated from its mesenchyme. Both tissues were treated separately with the carcinogens or with noncarcinogenic hydrocarbons (perylene or fluorene) in organ culture. Mesenchyme treated with the carcinogens was associated with freshly dissected, normal epithelium and cultivated on a hydrocarbon-free nutrient medium. Treated epithelium was associated in the same way with normal mesenchyme. Recombination experiments demonstrated that mesenchyme treated with the carcinogens allows bronchial tubulogenesis to occur. At the same time, associated epithelium responded to the influence of a treated mesenchyme with hyperplasia, pleomorphism, and sometimes invasive characteristics. Carcinogen-treated mesenchyme maintained its morphological role although it lost its control on epithelial proliferation. Epithelia directly treated with carcinogenic hydrocarbons had a lower incidence of pleomorphism; its associated mesenchyme remained unaltered. The changes in epithelial growth patterns are interpreted as the indirect response of epithelium to a mesenchyme altered by carcinogens. These results suggest that changes induced in mesenchyme by these compounds could play an important role in epithelial transformation.

2522 DNA CONTENT IN ATYPICAL GLIO-MESENCHYMAL CELLS DURING CHEMICAL CARCINOGENESIS IN THE ALBINO RAT CEREBELLUM. (Eng.) Kazantseva, I. A. (Acad. Medical Sciences USSR, Moscow, USSR); Yablonovskaya, L. Y. *Bull Exp Biol Med* 80(10): 1237-1240; 1975.

The DNA content in atypical glio-mesenchymal cells around a villous capsule surrounding a DMBA pellet in the cerebellum of seven albino SHK rats was determined. The results indicated that when carcinogenesis turns toward malignancy in the early stages, the rat cerebellum is characterized by a well-marked diploid modal class. Only in the case of microglioblastoma could a considerable degree of aneuploidy and polyploidy be found. These results indicated the comparatively low level of DNA synthesis in glial and connective tissue cells of the brain during a chronic inflammation around a foreign body.

2523 ANALYSIS OF THE EPOXIDE OF 7,12-DIMETHYLBENZ(A)ANTHRACENE AND ITS HYDROLYSIS PRODUCTS BY GAS CHROMATOGRAPHY. (Eng.) Morreal, C. E. (Roswell Park Memorial Inst., Buffalo, NY 14203); Dao, T. L.; Spiess, A. J. *Anal Biochem* 71(1): 125-132; 1976.

Column chromatography and electron-capture gas chromatography were used in the separation and quantitative analysis of the K-region epoxide of 7,12-dimethylbenz(a)anthracene, 5-hydroxy-7,12-dimethylbenz(a)anthracene, and trans-5,6-dihydro-5,6-dihydroxy-7,12-dimethylbenz(a)anthracene. Deactivation of chromatographic alumina with water permitted quantitative elution of the three compounds. In a solution of acetone and water, the epoxide was hydrolyzed to the 5,6-diol, but none of the 5-hydroxy derivative was formed.

2524 A RELIABLE, SENSITIVE, AND CONVENIENT RADIOACTIVE ASSAY FOR BENZPYRENE MONOOXYGENASE. (Eng.) DePierre, J. W. (Avdelningen for Biokemi, Stockholms Universitet, Fack, S-104 05 Stockholm, Sweden). *Anal Biochem* 63:470-484; 1975.

A new radioactive assay for 3,4-benzpyrene monooxygenase using tritiated substrate is described and compared with the commonly used fluorescent assay. The new procedure is based on a single extraction of the reaction mixtures with hexane after the reaction is stopped with 0.5 N NaOH in 80% ethanol, and the distribution of radioactivity is determined in the two phases. Approximately 7% of the reaction products are present in the hexane phase along with unreacted substrate. The assay indicated linearity of the reaction rate up to 20 min (longer with slower reactions) over a range of microsomal protein of 0-0.55 mg. Lineweaver-Burke analysis indicated a Km for benzpyrene of 22 μM. Comparison of ^{14}C- and ^{3}H-labeled substrates indicated that displacement of tritium into the water should not be a factor. Compared to the fluorescent assay, the new method gave enzyme activities that were approximately twice as high. Enzyme inhibition with cytochrome c, SKF 525-A, carbon monoxide, and anaerobiosis was similar with this assay and the fluorescent method. Enzyme activity in the liver microsomes of rats treated with methylcholanthrene was 10 times normal. The assay can be scaled down so that it is at least as sensitive as the fluorescent assay.

2525 AN IMPORTANT DIFFERENCE IN THE URINARY METABOLITES FORMED FROM 15,16-DIHYDROCYCLOPENTA(A)PHENANTHREN-17-ONE AND ITS CARCINOGENIC 11-METHYL HOMOLOGUE IN THE RAT. (Eng.) Coombs, M. M. (Imperial Cancer Res. Fund, Lincoln's Inn Fields, London, WC2A 3PX, England); Crawley, F. E. H. *Cancer Biochem Biophys* 1(3): 157-162; 1975.

Because polycyclic hydrocarbon carcinogens apparently require metabolic activation to a ''proximate'' form, the metabolic patterns of 15,16-dihydrocyclopenta(*a*)phenanthren-17-one (Ia) and its carcinogenic 11-methyl homolog (Ib) were compared by a chromatographic survey to find differences that would explain their differing biological activities. Metabolites were recovered by solvent extraction from the urine of rats that had been injected with Ia and Ib, and were compared by thin-layer chromatography (TLC). Also, the major urinary metabolite of the carcinogen was isolated chemically pure in the crystalline form. Patterns of the metabolites from each compound were similar, but there were important quantitative differences. Two phenols accounted for 30%-40% of the metabolites from the noncarcinogen, but only 10% from the carcinogen. The major metabolite of the latter had the novel dibenzoxepin structure 8,9-epoxy-1α,2β,15-ξ-trihydroxy-11-methyl-8,9-secogona-3,5,7,9,11,13-hexaen-17-one (XIV) and accounted for about 50% of the metabolites from this ketone. Small amounts of its dehydration product 8,9-epoxy-2,15-ξ-dihydroxy-11-methyl-8,9-secogona-1,3,5,7,9,11,13-heptaen-17-one (XIII) and of 15-16-dihydro-16-hydroxy-11-methylcyclopenta(a)phenanthren-17-one (IIb) also occurred in the extract from carcinogen-treated rats, but analogous structures were absent from the noncarcinogen.

2526 PREFERENTIAL INHIBITION OF THE SYNTHESIS OF 45S RIBOSOMAL RNA PRECURSOR BY N-ACETOXYACETYLAMINOFLUORENE IN RAT LIVER EPITHELIAL CULTURES. (Eng.) Kaplan, L. A. (Columbia Univ. Coll. Physicians and Surgeons, New York, NY 10032); Weinstein, I. B. *Chem Biol Interact* 12(1): 99-108; 1976.

Studies were conducted on the inhibitory effect of the carcinogen *N*-acetoxy-2-acetylaminofluorene (N-acetoxy-AAF) on the synthesis of RNA in cultured rat liver epithelial cells. A line of cells (K-16) was exposed for 10 min to increasing concentrations (0.2-1.5 μg/ml) of N-acetoxy-AAF. The cells showed a progressive decrease of [^{3}H]uridine incorporation into acid-precipitable material during a subsequent 10-min labeling period. Following a 10-min exposure to 0.5 μg/ml of N-acetoxy-AAF, the maximum inhibition of [^{3}H]uridine incorporation occurred 20 min after the removal of the carcinogen, and significant inhibition persisted for at least 45 hr. Concentrations of 0.5-1.0 μg/ml also caused a 50% inhibition of cloning efficiency of the K-16 cell line. Attempts were made to determine whether the extent of inhibition of RNA synthesis by N-acetoxy-AAF was similar for both 45S and hnRNA. Data indicated that only the synthesis of the 45S RNA peak was significantly reduced by the N-acetoxy-AAF treatment. The 45S RNA activity was reduced 42% after N-acetoxy-AAF treatment, while the specific activities in the hnRNA regions of the gel were reduced much less: 3% for the >45S, 15% for the <45S-28S, and 0% for the 28S-18S RNA regions. Thus, the synthesis of 45S RNA is preferentially inhibited by N-acetoxy-AAF. This preferential inhibition is not explained adequately by invoking the high guanine-cytosine content of these cistrons. Nor does it appear that the large target size of rRNA cistrons is responsible. The preferential inhibition may result from certain regions of the DNA being more susceptible to chemical modification due to local secondary or tertiary structure of that region of the DNA. Also, the presence or absence of specific chromosomal proteins may render specific regions of the DNA more susceptible to chemical modification. The inhibition could also result from the responses of different classes of DNA-dependent RNA polymerases to chemical modification of DNA.

2527 SYNTHESIS OF AZIRIDINES. (Fre.) Girault, Y. (Laboratoire de Chimie Physique Organique, Universite de Nice, Campus Valrose, 06034 Nice Cedex, France); Decouzon, M.; Azzaro, M. *Tetrahedron Lett* (15): 1175-1176; 1976.

The authors present a simple, rapid method for the synthesis of secondary aziridines through the action of an aluminum reducing agent [redal, $NaAlH_2(OCH_2CH_2CH_3)_2$] on the derivatives of cyclic ketones (either saturated or not substituted by an aromatic grouping), oximes, and hydrazone iodides. This method was applied to a series of cyclohexanones and to camphor. It was valid only for oximes carrying a substituent larger than a methyl group. Their yields varied from 56% to 66%. The reduction of iodides produced a constantly high yield that ranged from 46% to 85%. Access to cis-substituted secondary aziridines is possible through the action of an aluminum reduction agent on derivatives of saturated ketones (oximes and hydrazone iodides). (6 references)

2528 ACUTE TOXICITY OF DIMETHYLNITROSAMINE IN THE PRESENCE OF INHIBITORS OF DMN DEMETHYLASE. (Eng.) Friedman, M. A. (Med. College Virginia, Virginia Commonwealth Univ., Richmond, VA 23298); Sanders, V. *Experientia* 32(4): 495-496; 1976.

The acute toxicity of dimethylnitrosamine (DMN) in the presence of inhibitors of DMN demethylase (piperonyl butoxide, dibutylnitrosamine, nitrososarcosine, and diethylnitrosamine) was tested in male Swiss albino mice. All injections were ip. Each of the compounds tested induced a greater than 50% inhibition of DMN demethylase and suppressed the mutagenic action of DMN. However, only the diethylnitrosamine affected the LD_{50} of the DMN. All mice given 100 mg/kg dimethylnitrosamine and 10.7 mg/kg DMN died. The results suggest that DMN demethylase may not be involved in the acute toxicity of DMN.

2529 NONRANDOM NATURE OF IN VIVO METHYLATION BY DIMETHYLNITROSAMINE AND THE SUBSEQUENT REMOVAL OF METHYLATED PRODUCTS FROM RAT LIVER CHROMATIN DNA. (Eng.) Ramanathan, R. (Frederick Cancer Res. Center, Post Box ''B'', Frederick, MD 21701); Rajalakshmi, S.; Sarma, D. S. R.; Farber, E. *Cancer Res* 36(6): 2073-2079; 1976.

The intragenomic distribution of alkylated products and the rate of their subsequent removal from liver chromatin DNA following administration of the hepatocarcinogen dimethylnitrosamine (DMN) were studied to determine whether methylation of liver chromatin DNA by DMN and the subsequent in vivo removal of DNA-bound methylated products are random. Liver chromatin DNA was fractionated into nuclease-digestible and nondigestible material 4 hr after administration of ^{3}H-DMN (0.5 mg/250 μCi/100 g body weight). Digestion of the methylated liver chromatin with pancreatic DNase I or micrococcal nuclease and analysis of nuclease-digested acid-soluble products revealed a discrepancy between the radioactivity released (72%) and the nucleotides released (50%), as measured by the absorbance at 260 nm. This discrepancy disappeared, and the rate and extent of release of both the radioactivity and the absorbance at 260 nm were identical when the total purified DNA isolated from methylated chromatin was used as the substrate instead of chromatin DNA in the nuclease reaction. These results, together with the fact that the guanine contents of the DNA of the two fractions of chromatin were identical, suggest that methylation of the nuclease-accessible region of hepatic chromatin DNA is relatively greater than that of the inaccessible region. Removal of methylated products in the accessible region of the chromatin DNA further revealed that, of the methylated products present at 4 hr, 62% is lost by 3 days, 87% is lost by 1 wk, and 94% is lost by 2 wk. Loss from the nuclease-inaccessible region of chromatin DNA is, however, only 27% by 3 days, 49% by 1 wk, and 86% by 2 wk, which suggests that the removal of methylated products from this region is relatively slower compared to the accessible region. It is concluded that the distribution and removal of DMN-induced methylated products in liver chromatin DNA is nonrandom, as measured by this technique.

2530 EVALUATION OF GENETIC RISKS OF ALKYLATING AGENTS. II. HAEMOGLOBIN AS A DOSE MONITOR. (Eng.) Osterman-Golkar, S. (Wallenberg Lab., Univ. Stockholm, Lilla Frescati, S-104 05 Stockholm 50, Sweden); Ehrenberg, L.; Segerback, D.; Hallstrom, I. *Mutat Res* 34(1): 1-10; 1976.

The authors determined the degree of hemoglobin alkylation at different times after treating male CBA mice with a directly active alkylating agent, ethylene oxide, and an agent that requires metabolic activation, dimethylnitrosamine. The compounds were administered ip (0.5 ml saline/25 g body weight). Because of the random alkylation of RBC of various ages and the stability of alkylated hemoglobin, the amount of alkylated amino acids in hemoglobin decreases linearly with time, reaching the value zero after about 40 days, the life span of erythrocytes in the mouse. This provides a basis for the use of hemoglobin as a monitor for integral doses of genotoxic environmental chemicals. Since the lifetime of hemoglobin is not affected by alkylation, this method may be used to integrate the dose and the risk for a fairly long time, comparable with the lifetime of erythrocytes in man (about 4 mo), provided the reaction products are chemically stable. (16 references)

2531 TRANSPLACENTAL EFFECTS OF NITROSAMINES IN SYRIAN HAMSTERS: I. DIBUTYLNITROSAMINE AND NITROSOHEXAMETHYLENEIMINE. (Eng.) Althoff, J. (Univ. Nebraska Medical Center, 42nd and Dewey Ave., Omaha, NB 68105); Pour, P.; Grandjean, C.; Eagen, M. *Z Krebsforsch* 86(1): 69-75; 1976.

The transplacental carcinogenic effects of dibutylnitrosamine (DBN) and nitrosohexamethyleneimine (N-6-MI) were examined in Syrian hamsters to demonstrate whether or not the compounds pass the placenta and to determine the possible effect on the first generation when the parents were treated at noncarcinogenic dose levels. A proportion of both substances reached the fetal tissue unaltered. No macroscopic malformations were observed in the offspring; however, postnatal mortality was high. Respiratory tumors were found upon histologic examination of surviving animals. Single doses of 30 mg/kg body weight DBN and two doses of 10 mg/kg N-6-MI did not induce tumors in the P-generation, but led to a low tumor incidence in the F_1-generation (DBN, 7.0%; N-6-MI, 2.0%). Treatment for up to 8 days during the second half of pregnancy led to a higher tumor incidence in the P-generation (DBN, 22%; N-6-MI, 20%), than in the F_1-generation (DBN, 6.0%; N-6-MI, 10%). These results indicate that transplacental carcinogenesis may be an established method of tumor induction in different species by various groups of carcinogens.

2532 PHYSICOCHEMICAL ALTERATIONS IN THE CONFORMATION OF RAT LIVER CHROMATIN INDUCED BY CARCINOGENS IN VIVO.

(Eng.) Nicolini, C. (Temple Univ. Health Sciences Center, Philadelphia, PA 19140); Ramanathan, R.; Kendall, F.; Murphy, J.; Parodi, S.; Sarma, D. S. R. *Cancer Res* 36(5): 1725-1730; 1976.

Administration of methyl methanesulfonate (120 mg/kg), dimethylnitrosamine (5 mg/kg), or methylnitrosourea (80 mg/kg) to male Wistar rats resulted in an increased ellipticity in circular dichroism spectra and in an enhanced ability to bind ethidium bromide in the liver chromatin. Although shearing of the chromatin preparations increased both the ellipicity and number of binding sites for ethidium bromide, the carcinogen-induced effects were noticeable whether or not chromatin was sheared. Although the doses of the carcinogens used in these studies were equivalent in ability to induce strand breaks in liver DNA at 4 hr, their effects on the induction of conformational changes in liver chromatin were different. For example, methyl methanesulfonate induced the minimum conformational changes in liver chromatin at 4 hr, whereas methylnitrosourea induced the maximum changes at 4 hr. Methylmethanesulfonate and dimethylnitrosamine, on the other hand, induced maximum changes at 3 days. The conformational changes induced by methyl methanesulfonate and methylnitrosourea, and not by dimethylnitrosamine, tended to be repaired by 14 days. The results indicate that methylating carcinogens induce alterations in the conformation of rat liver chromatin.

2533 THE EFFECTS OF SOME CARCINOGENIC NITROSO COMPOUNDS ON THE RAT LIVER NUCLEOLUS.

(Eng.) Terao, K. (Res. Inst. Chemobiodynamics, Chiba Univ. Narashino 275, Chiba, Japan). *Z Krebsforsch* 86(1): 47-54; 1976.

The effects of eight carcinogenic nitroso compounds (dimethylnitrosamine, diethylnitrosamine, butylnitrosamine, methylnitrosourea, butylnitrosourea, N-methylnitrosourethane, N-butylnitrosourethane, and N-methyl-N-nitro-N-nitrosoguanidine) on the rat liver nucleus were characterized and compared with those of five corresponding nonnitroso compounds (butylamine, methylurea, butylurea, N-butylurethane, and dimethylamine-HCl). A total of 150 male Wistar rats (90-110 g), divided into groups of 10-20, were given the compounds in aqueous or dimethylformamide solution by stomach tube; tissue was removed from the left lateral lobe of the liver for electron and light microscopy. The nucleoli of the rats underwent a variety of specific and nonspecific alterations. After administration of dimethylnitrosamine (25 mg/100 g) and diethylnitrosamine (200 mg/100 g), an unusual segregation of the nucleolus was encountered in the cells with karyorrhexis. Most characteristic of the segregation were the scattered ribosomelike granules at the outermost layer of the altered nucleolus. Frequently, microspherules with halos 150 nm in diameter were found in all rats treated with the nitroso compounds, which were reported to be carcinogenic in organs other than the liver. With the exception of butylurea and butylamine, no such specific microspherules were found in the nucleoli after corresponding nonnitroso compounds were administered.

2534 LIFE-SPAN INVESTIGATIONS FOR CARCINOGENICITY OF SOME IMMUNE-STIMULATING, IMMUNODEPRESSIVE AND NEUROTROPIC SUBSTANCES IN SPRAGUE-DAWLEY-RATS.

(Eng.) Schmaehl, D. (Institut fuer Toxikologie und Chemotherapie am Deutschen Krebsforschungszentrum, D-6900 Heidelberg, Im Neuenheimer Feld 280, W. Germany); Habs, M. *Z Krebsforsch* 88(1): 77-84; 1976.

The authors evaluated the chronically toxic effects of immune-stimulating, immunodepressive, or neurotropic substances on the chemical carcinogenesis induced by ethylnitrosourea (ENU). These compounds were administered during the period of ENU tumor induction. A total of 858 12-day-old Sprague-Dawley rats of both sexes were given 5% of the acute LD_{50} per week of complete Freunds' adjuvant, bacille Calmette Guerin (BCG) vaccine, human albumin, vitamin A acid, rabbit antirat lymphocytic serum, hydrocortisone, amethopterin, cyclophosphamide, atropine, pilocarpine, nicotine, and phenobarbital. Complete Freunds' adjuvant, an immune-stimulating substance, decreased the lifetime of the animals (87 wk, vs. 94-96 wk in controls); 11 animals developed malignant tumors (7 adenocarcinomas of the mamma and 2 subcutaneous fibrosarcomas were found in 33 females). BCG vaccine also decreased the lifetime, but it, human albumin, and vitamin A acid were not carcinogenic (the last two compounds had no effect on survival time). Rabbit antirat lymphocytic serum, an immunodepressive agent, decreased survival significantly (81 wk, males; 82 wk, females), but was not carcinogenic. The known carcinogenic properties of cyclophosphamide were verified. The applied dose of this compound (4 mg/kg/wk) also reduced the mean survival times (87 wk, males; 85 wk, females), and a significantly increased tumor rate was observed (12/36 females and 5/32 males). Hydrocortisone and amethopterin had no carcinogenic effects. The neurotropic substances nicotine and phenobarbital diminished the mean survival times, but they and atropine and pilocarpine were not carcinogenic.

2535 TEMPORARY CELL CYCLE ARREST IN NEURAL AND EXTRANEURAL DEVELOPING RAT TISSUES AFTER EXPOSURE TO METHYL- AND ETHYLNITROSOUREA.

(Eng.) Bosch, D. A. (Dept. Pathology, Univ. Groningen, Oostersingel 63, Groningen, Holland); Ebels, E. J. *Z Krebsforsch* 86(1): 23-31; 1976.

To establish whether inhibition of mitototic activity can be explained by an arrest of the cell cycle in, or before entering into, the S phase and to find a time correlation between re-entrance into the S phase and subsequent reappearance of mitototic figures, the cycle arrest of mitotically inhibited rat cells was assessed by pulse labeling, with tritiated thymidine, and by autoradiography. Eight-day-old rats were exposed to 20 or 100 mg/kg body weight of either methylnitrosourea (MNU) or ethylnitrosourea (ENU), followed by injection of 10 μCi/g body weight of (^{3}H-methyl)-thymidine. A 100-mg dose of MNU in both neural and extraneural tissues caused a longer total inhibition of S phase radioactivity than the same dose of ENU. Reappearance of S phase cells in the neural tissues occurred later (36-48 hr) than in the extraneural tissues (24-36 hr) for both drugs. In both neural and extraneural tissues, S phase cells reappeared about 12 hr earlier than M phase cells. After a 20-mg dose of MNU or ENU in both neural and extraneural tissues there was a clear decrease in S phase radioactivity after only a 6-hr interval. In developing rat tissues, mitototic activity and S phase radioactivity decreased with increasing single doses of MNU or ENU. The results indicate an arrest in or before the cells entered the S phase. Recurrence of DNA synthesis and re-entrance of damaged cells into their cycle before the altered bases are eliminated from DNA may be of importance in oncogenesis.

2536 THE EFFECT OF HORMONE INDUCED STRESS UPON THE EXTENT OF ALKYLATION OF RAT LIVER NUCLEIC ACIDS BY N-METHYL-N-NITROSOUREA. (Eng.) Magin, M. N. (Christie Hosp. and Holt Radium Inst., Manchester M20 9BX, England); O'Conner, P. J.; Craig, W. A. *J Krebsforsch* 84(3): 217-222; 1975.

To assess the possible role of induced stress in carcinogenesis, the effect of stress was evaluated on DNA synthesis in the resting liver and on the initial reaction of an ultimate carcinogen with cellular macromolecules with use of male Wistar rats. Methyl methanesulfonate (MMS, 50 mg/kg, ip), N,N-dimethylnitrosamine (DMN, 2 mg/kg, ip), and N-methyl-N-nitrosourea (MNU, 30 mg/kg, ip) each resulted in an initial (30 min) stimulation of thymidine incorporation (approximately 25%-55%), followed by a suppression of thymidine incorporation at 2-4 hr after injection (25%-50%). Hormone treatment (adrenalin, 100 μg/kg, im plus hydrocortisone, 10 mg/kg, im) resulted in progressive inhibition to approximately 55% at 2 hr with no further change at 4 hr. Higher levels of DMN (10-30 mg/kg) resulted in immediate suppression of DNA synthesis. With higher levels of MMS (100 mg/kg), there was still an initial rise in DNA synthesis, but the ensuing fall was more rapid and more profound. When the extent of alkylation of DNA was assessed in terms of the percentage of 7-methylguanine, treatment with stress hormones increased the degree of alkylation of both DNA and tRNA by MNU in a dose-dependent fashion (34%-52% increased alkylation). Possible mechanisms of action are discussed.

2537 STRAND BREAKAGE IN RAT BRAIN DNA AND ITS REPAIR INDUCED BY ETHYLNITROSOUREA IN VIVO. (Eng.) Hadjiolov, D. (Regeneration Res. Lab., Bulgarian Acad. Sciences, Sofia, Bulgaria); Venkov, L. *Z Krebsforsch* 84(3): 223-225; 1975.

The effect of a single carcinogenic dose of ethylnitrosourea (20 mg/kg ip) on the initial damage of brain DNA and its repair was studied in 7-day-old rats. The brain DNA was labeled by injection of ^{3}H-thymidine. The single dose of ENU was highly damaging to the DNA, as shown by sedimentation in alkaline sucrose gradients. At the fourth hour single-strand breaks were induced. By 24 hr after treatment there was a shift toward the heavier fractions of the gradient, but large fractions of radioactivity remained maximally displaced from the control position, showing that repair had not been completed. The induction of these single-strand breaks and their incomplete repair might be an important factor in brain carcinogenesis.

2538 GROWTH SUPPORT OF SMALL B16 MELANOMA IMPLANTS WITH NITROSOUREA-STERILIZED FRACTIONS OF THE SAME TUMOR. (Eng.) Dykes, D. J. (Kettering-Meyer Labs., Southern Res. Inst., Birmingham, AL 35205); Griswold, D. P., Jr.; Schabel, F. M., Jr. *Cancer Res* 36(6): 2031-2034; 1976.

To study chemically induced growth-supporting effect (GSE) and its possible influence on chemotherapy, B16 melanoma cells (from the ear of a C57BL/6J mouse) sterilized in vitro with 1,3-bis(2-chloroethyl)-1-nitrosourea or in vivo with trans-1-(2-chloroethyl)-3-(4-methylcyclohexyl)-1-nitrosourea were used to enhance the percentage of tumor takes with small sc implants of viable cells and to reduce the latent period between tumor implantation and palpability. The admixture of trans-1-(2-chloroethyl)-3-(4-methylcyclohexyl)-1-nitrosourea-inactivated cells with viable cell implants reduced the number of cells required to produce tumors in 50% of the animals by approximately 3 $\log_{10}$ units and markedly reduced the time of tumor appearance from implants of up to 10^6 cells. Similar results were obtained with 1,3-bis(2-chloroethyl)-1-nitrosourea-sterilized cells. The GSE obtained with the nitrosourea-inactivated cells appeared to be as pronounced as that previously reported, for this tumor system, with radiation-inactivated cells.

2539 CARCINOGENIC EFFECT OF N-BUTYL-N-NITROSOURETHAN ON CDF$_1$ MICE. (Eng.) Fujii, M. (Sch. Medicine, Tokai Univ., Boseidai, Isehara 259-11, Japan). *Gann* 67(2): 231-239; 1976.

The author studied the induction of tumors in mice by N-butyl-N-nitrosourethan (BNUR). Oral administration of 0.04% BNUR in deionized water to CDF$_1$ mice for 20 wk produced papillomas in the esophagus and forestomach of all 58 mice; and 24 had squamous cell carcinoma. No skin tumors developed when 0.1%, 0.5%, or 1% BNUR in acetone was painted on the backs of CDF$_1$ mice three times a week for 25 wk. No sarcomas developed within 30 wk when a dose of 10, 60, or 120 mg/ml of BNUR in dimethyl sulfoxide solution was injected sc into the backs of CDF$_1$ mice, 10 times at 1-wk intervals. In the in vitro malignant transformation test, in which golden hamster lung fibroblast cells were treated with BNUR at a final concentration of 30 and 60 μg/ml for 24 hr, malignant transformation did not occur during the 150-day observation period. The carcinogenicity and possible mode of action of BNUR are discussed on the basis of these results. (13 references)

2540 CYTOGENETIC STUDY OF DDT ON HUMAN LYMPHOCYTES IN VITRO. (Eng.) Lessa, J. M. M. (Centro de Ciencias Biologicas, Universidade Federal de Alagoas, Brazil); Becak, W.; Rabello, M. N.; Pereira, C. A. B.; Ungaro, M. T. *Mutat Res* 40: 131-138; 1976.

To investigate the in vitro cytogenetic effects of DDT on human chromosomes, short-term lymphocyte cultures were exposed to DDT dissolved in acetone and diluted in water or to DDT dissolved in acetone and added directly to the culture (10^{-4} and 2×10^{-3} ml/ml medium, respectively). Controls received the same volume of acetone. Cells were scored for aneuploidy, polyploidy, and structural aberrations. No correlation was found between DDT doses and cells with chromosomal aberrations. The Poisson test of comparison between means showed that at certain DDT concentrations (0.20, 4.05, and 8.72 μg/ml) the proportion of cells with structural aberrations was significantly greater than in the controls.

2541 VINYL CHLORIDE AND THE PRODUCTION OF PVC. (Eng.) Barnes, A. W. (ICI Plastics Div., Welwyn Garden City, Hertfordshire, England). *Proc R Soc Med* 69(4): 277-281; 1976.

The polymerization characteristics of vinyl chloride are described, and the process for producing polyvinyl chloride (PVC) is outlined. Interfaces of exposure of humans to PVC are outlined, and theoretical exposure levels for workers at various stages in the production process and for the average U.K. civilian are calculated. Average annual dietary ingestion is calculated as 0.0001 g/yr. Atmospheric exposure for polymerization workers has decreased from approximately 1,000 ppm in 1940 to approximately 5 ppm in 1975, although certain workers might have been exposed to as much as 3,000 ppm in 1940. The daily dose of the polymerization plant worker who had been exposed to 1,000 ppm is calculated at 0.36 g/kg, the dose for the polymerization plant worker presently exposed to 5 ppm is 0.0018 g/kg, and that of the average citizen 0.000000004 g/kg.

2542 PRELIMINARY RESULTS OF GREY-SCALE ULTRASONOGRAPHY IN THE DETECTION OF VINYL CHLORIDE RELATED LIVER AND SPLEEN DISEASE. (Eng.) Taylor, K. J. W. (Yale Univ. Sch. Medicine, New Haven, CT); Barrett, J. J.; Williams, D. M. J.; Smith, P. M.; Duck, B. W. *Proc R Soc Med* 69(4): 292-295; 1976.

In an attempt to devise a method for mass examination of vinyl chloride-exposed workers, a pilot project was carried out with grey-scale ultrasonography, a technique that allows normal tissue parenchyma to be displayed. Results of tests with 19 volunteers exposed to vinyl chloride suggest that this method is a complement to rather than a replacement for current techniques. The main advantage is that it is a noninvasive method of monitoring populations without exposing them to ionizing radiation.

2543 EVALUATION OF THE GENETIC EFFECTS INDUCED BY VINYL CHLORIDE MONOMER (VCM) UNDER MAMMALIAN METABOLIC ACTIVATION: STUDIES IN VITRO AND IN VIVO. (Eng.) Loprieno, N. (Istituto di Genetica, University, Pisa, Italy); Barale, R.; Baroncelli, S.; Bauer, C.; Bronzetti, G.; Cammellini, A.; Cercignani, G.; Corsi, C.; Gervasi, G.; Leporini, C.; Nieri, R.; Rossi, A. M.; Stretti, G.; Turchi, G. *Mutat Res* 40: 85-95; 1976.

The mutagenic activity of vinyl chloride monomer (VCM) was observed on yeast in the presence of a pure preparation of mouse (Swiss albino) liver microsomes and in the ''host-mediated assay''; the gene conversion inducible by *Saccharomyces cerevisiae* was also observed. VCM in the presence of purified microsomes (sedimented at 105,000 *g*) was converted into an active metabolite(s) that produced gene mutations in the yeast *S. pombe* (forward mutation) and gene conversions in two loci of diploid *S. cerevisiae*. No mutagenic activity was found when yeast cells were treated with VCM, a phosphate buffer, or microsomes alone; this demonstrates that the enzyme necessary to metabolize VCM into a biologically reactive compound is not present in the yeast. Moreover, the compound was active in the host-mediated assay, when mice were treated with an oral dose of 700 mg/kg of VCM. These results have demonstrated that VCM is mutagenic for eukaryotic organisms and that it produces other genetic changes; this activity is liver-microsome dependent and dose dependent. It is recommended that reliable methodologies, like this mammalian metabolic activation one, for assessing the toxicological values of many industrial compounds before they reach production, be made available.

2544 EXPERIMENTAL INVESTIGATIONS ON THE INFLUENCE UPON THE CHEMICAL CARCINOGENESIS. THIRD COMMUNICATION: STUDIES WITH 1,2-DIMETHYLHYDRAZINE. (Eng.) Schmaehl, D. (Institut fuer Toxikologie und Chemotherapie am Deutschen Krebsforschungszentrum, D-6900 Heidelberg, Im Neuenheimer Feld 280, W. Germany); Danisman, A.; Habs, M.; Diehl, B. *Z Krebsforsch* 86(1): 89-94; 1976.

To determine whether chemical carcinogenesis can be influenced by simultaneous applications of immunostimulating or immunodepressive agents, 1,2-dimethylhydrazine (DMH) was injected sc monthly into 576 2-day-old Sprague-Dawley rats; the effects of high cholesterol and vegetarian diets and enzyme-stimulating substances on tumor development were also studied. After 10 monthly injections of 30 mg/kg of DMH to the rats, malignant tumors were found in about 90% of the animals after a mean induction period of 330 days. Of the tumors induced, 72% were formed in the colon, 17% were squamous cell carcinomas of the ear duct, 13% were adenosarcomas of the kidney, and 11% were hepatocellular carcinomas. Additional treatment with immunodepressive compounds, (cyclophosphamide, methotrexate, hydrocortisone), immunostimulating substances (bacille Calmette Guerin, albumin, vitamin A acid), and an enzyme-stimulating agent (Luminal) did not alter incidences and induction periods of tumors. The vegetarian diet group had a significant prolongation of induction (about 392 days). These rats also had fewer liver and kidney tumors. These studies demonstrate that DMH is not only a carcinogen to the intestine but also to the ear duct, kidney, and liver of the rat. No interpretation as to the significance of the vegetarian diet was made.

2545 MUTAGENIC EFFECT OF SODIUM NITRITE ON CULTURED MOUSE CELLS. (Eng.) Kodama, F. (Yokohama City Univ. Sch. Medicine, Urafune-cho, Minami-ku, Yokohama 232, Japan); Umeda, M.; Tsutsui, T. *Mutat Res* 40: 119-124; 1976.

The direct mutagenic and other biological effects of sodium nitrite on FM3A cells (a C3H mammary carcinoma cell line) were studied. The chromosomal preparations revealed that severe aberrations were induced in more than 80% of the mitotic plates at 10^{-2} M and in nearly 40% at $10^{-2.5}$ M after 24 and 48 hr of treatment. According to the results of alkaline sucrose gradient analysis, sedimentation profiles of single-strand cell DNA treated at as high as 10^{-1} M for 24 hr scarcely changed from that of control cell DNA. Induction of 8-azaguanine-resistant mutation was demonstrated above 10^{-3} M sodium nitrite.

2546 CYTOTOXICITY OF HAIR COLOURANT CONSTITUENTS: CHROMOSOME DAMAGE INDUCED BY TWO NITROPHENYLENEDIAMINES IN CULTURED CHINESE HAMSTER CELLS. (Eng.) Kirkland, D. J. (Inst. Center Res., Chalfont St. Giles, Bucks, England); Venitt, S. *Mutat Res* 40(1): 47-56; 1976.

Two aromatic amines, 2-nitro-p-phenylenediamine (4NPPD) and 4-nitro-o-phenylenediamine (4NOPD) were cytotoxic (in the dose-range 5-100 μg/ml) to CHMP/E cells (a recently derived near-diploid (2n = 24) clone of Chinese hamster prostate cells) following 5 days continuous exposure. 2NPPD was more toxic than 4NOPD at equivalent doses. These cytotoxic effects were paralleled by a time-dependent increase in the yield of chromosome aberrations following exposure of CHMP/E cells to 25 μg/ml 2NPPD or 4NOPD, compared with control cultures. The incidence of damaged metaphase exchanges after 7 days' exposure was 26% for 4NOPD, 40% for 2NPPD, and 4% for controls. The use of 2NPPD and 4NOPD as hair-colorant constituents may be associated with a long-term health hazard.

2547 THE DIFFERENTIAL EFFECTS OF CHEMICAL CARCINOGENS ON VITAMIN A STATUS AND ON MICROSOMAL DRUG METABOLISM IN NORMAL AND VITAMIN A-DEFICIENT RATS. (Eng.) Hauswirth, J. W. (Sinai Hosp. Baltimore, Inc., Baltimore, MD 21215); Brizuela, B. S. *Cancer Res* 36(6): 2031-2034; 1976.

To determine whether an interrelationship exists between the effects of vitamin A and chemical carcinogens on hepatic microsomal cytochrome P-450, male Sprague-Dawley rats were maintained on a vitamin A-deficient diet for 5 wk. Although serum and hepatic levels of vitamin A were significantly lower, no outward signs of vitamin A deficiency were present. Hepatic microsomal levels of cytochrome P-450 in the vitamin A-deficient animals were 70% that of the control animals. Of the three microsomal enzymes studied, ethylmorphine N-demethylase, aniline hydroxylase, and aminopyrine N-demethylase, only the last one was adversely affected by vitamin A deficiency. 3-Methylcholanthrene, phenobarbital, and 2-acetylaminofluorene had a greater inductive effect on cytochrome P-450 in vitamin A-deficient rats. 4-Dimethylaminoazobenzene treatment decreased the level of cytochrome P-450 in control rats more than in deficient rats. The hepatic concentration of vitamin A was significantly reduced in control rats that were given injections of 3-methylcholanthrene, 2-acetylaminofluorene, or phenobarbital. Benzo(a)pyrene and 4-dimethylaminoazobenzene had less effect.

2548 LIGHT- AND ELECTRON MICROSCOPIC STUDIES OF THE RENAL CARCINOMA INDUCED IN SYRIAN HAMSTERS BY DIETHYLSTILBESTROL. (Eng.) Hara, Y. (Yamaguchi Univ., Sch. Medicine, Ube, Japan). *Bull Yamaguchi Med Sch* 22(1): 49-62; 1975.

Diethylstilbestrol (DES)-induced renal carcinoma of male Syrian golden hamsters was studied by light and electron microscopy. The significance of the changes observed is discussed in relation to ''dis-differentiation'' of neoplastic cells. Fifteen hamsters (6-7 wk old) were given 0.6 mg DES in 0.2 ml sesame oil sc qod, were killed on day 200, 250, 300, or 350, and their kidney specimens processed for electron microscopy. Two methods of fixation were used: (a) the animals were decapitated, and small pieces of neoplastic kidney were fixed in glutaraldehyde, and postfixed in osmium tetroxide; and (b) the animals were anesthetized with ether, the kidney was perfused in situ with glutaraldehyde and then removed, and well-fixed areas were cut into 1 mm cubes, which were again fixed in glutaraldehyde and postfixed in osmium tetroxide. Semithin sections from the blocks for electron microscopy were examined by light microscopy after toluidine blue staining. Ultrastructural features that appeared to be specific to this tumor were clusters of fibrillar material, intranuclear bodies, and intracytoplasmic lumina with cilia and microvilli. Swelling of mitochondria and paucity of cristae were observed and various forms of abnormal mitochondria were seen. The endoplasmic reticulum was generally sparse and primitive in appearance. The Golgi apparatus was well developed in spite of marked cellular atypism. Renal tubular epithelium of Syrian hamster has no cilia except under pathological conditions. It is suggested that neoplastic transformation may be accompanied by the derangement of organ-specific or tissue-specific functions or structures and that non-specific phenotypes are manifested by the tumor cells through the unmasking of genes.

2549 STRUCTURAL AND FUNCTIONAL ABNORMALITIES IN THE SEX ORGANS OF MALE OFFSPRING OF MOTHERS TREATED WITH DIETHYLSTILBESTROL (DES). (Eng.) Gill, W. B. (Univ. Chicago Pritzker Sch. Medicine, IL 60637); Schumacher, G. F. B.; Bibbo, M. *J Reprod Med* 16(4): 147-153; 1976.

The in utero effects of diethylstilbestrol (DES) on the human male genital tract are reported in this followup study of male offspring of DES-treated mothers. Both anatomical and functional abnormalities were significantly greater in the DES-exposed males than in control males. The mothers were all participants in a prospective, randomized, double blind study of the effects of DES on pregnancy during the early 1950s. Epididymal cysts, hypotrophic testes, and capsular induration of the testes were the more common genital lesions found in 27% of 134 DES-exposed males compared to a 7% incidence in 119 controls. Spermatozoa analyses revealed severely pathologic changes (Eliasson score >10) in 29% of 28 DES-exposed males and 0% of 18 control males (with or without physical genital abnormalities). Abnormal findings on physical examination combined with sperm abnormalities (Eliasson score ≥5) were found in 29% of DES-exposed males versus 0% of 18 controls. Cytological examination did not reveal malignant cells from the following materials: urines before and after prostatic massage or ejaculation, prostatic fluids, or aspirates from epididymal cysts.

2550 ENDOMETRIAL ABNORMALITIES OCCURRING IN YOUNG WOMEN ON LONG-TERM SEQUENTIAL ORAL CONTRACEPTION. (Eng.) Lyon, F. A. (Univ. Minnesota Sch. Medicine, Minneapolis, MN); Frisch, M. J. *Obstet Gynecol* 47(6): 639-643; 1976.

Twelve patients, age 21-32 yr, using Oracon underwent evaluation of the endometrium because of the reported occurrence of endometrial cancer in young women on long-term sequential oral contraceptives. All patients were totally asymptomatic and had been using Oracon for 13-93 mo. Seven patients exhibited adenomatous endometrial hyperplasia of varying degrees of severity. Two patients developed severe atypical adenomatous hyperplasia bordering on in situ adenocarcinoma. There seems to be an association between long-term Oracon use and the development of these abnormalities. Ten of the 12 patients had exhibited oligomenorrhea prior to the beginning of Oracon therapy, but no endometrial tissue was obtained at that time. It is not known if these patients would have developed abnormalities had Oracon not been prescribed. Patients currently using Oracon should be observed carefully for signs of abnormal uterine bleeding. Even in the absence of symptoms, the endometrium should be periodically evaluated. All young patients who develop adenocarcinoma of the endometrium should be questioned about sequential oral contraceptive use to determine the true incidence of endometrial cancer development in patients using these contraceptives.

2551 INHIBITION OF PROLACTIN SECRETION BY ERGOT ALKALOIDS. (Eng.) Nasr, H. (Inst. Res. Human Reproduction, Ministry Health, Teheran, Iran); Pearson, O. H. *Acta Endocrinol (Kbh)* 80(3): 429-443; 1975.

The effects of various ergot alkaloids on prolactin (Pr) secretion in adult female rats were determined by radioimmunoassay. Ergocornine (ECO) and ergocristine (ECR) in doses of 0.25 to 1.0 mg lowered serum Pr markedly by 1 hr (with the effect persisting for 24 hr at the larger doses). Several other ergots had similar effects, while the dihydro derivatives had diverse responses. The pro-estrous surge of Pr could be blocked by ECR or ECO without interfering with the estrous cycle. ECO or ECR could suppress the rise in serum Pr induced by estradiol benzoate (5-50 μg) in oophorectomized rats. Perphenazine stimulation of Pr could be partially antagonized by ECO depending on dose and timing of injections. ECO (2 mg) produced an abrupt cessation of lactation with concomitant fall in serum Pr. Evaluation of pituitary Pr concentration in lactating and intact rats receiving ECO led to the conclusion that release of Pr from the pituitary was affected. ECO (1 or 2 mg) produced a regression of dimethylbenz(a)anthracene-induced rat mammary tumors.

2552 OESTROGENS AND THE ETIOLOGY OF BREAST CANCER: UTILIZATION OF DEMOGRAPHIC DIFFERENCES TO TEST A HYPOTHESIS. (Eng.) MacMahon, B. In: *Host Environment Interactions in the Etiology of Cancer in Man. Proceedings of a Meeting Held at Primosten, Yugoslavia, 27 August-2 September 1972.* Edited by R. Doll, I. Vodopija and W. Davis. International Agency for Research on Cancer. (Lyon, France): pp. 163-167; 1973.

The use of population comparisons to test the hypothesis that women who metabolize a low proportion of their ovarian estrogens to estriol (E3) are at high risk of breast cancer and the sub-hypothesis that the proportion of estrogen metabolized to estriol (E3) is particularly significant during the first half of reproductive life is illustrated. The hypothesis stems from the demonstration that the two other estrogen fractions, estrone and estradiol (E1 and E2, respectively) are carcinogenic but E3 is not. The most accurate measure of the proportion of estrogen metabolized to E3 is the ratio of E3 to the other two fractions in urine (E3/(E1 + E2) = estriol ratio). The first comparison of estrogen profiles was made between women from three areas in Asia and women from two North American cities, because the five- or six-fold difference in breast cancer rates between Asian and North American populations is one of the strongest epidemiological features of the disease. The results from a pilot study indicate that the estriol ratio is considerably higher for the Asian than for the North American women, especially in the 15-19 yr age group. Another investigation of estrogen profiles is being conducted among Caucasian, Chinese, and Japanese women in Hawaii, where the Chinese women now have breast cancer rates almost as high as the Caucasian, and where the Japanese, whose migration came later than that of the Chinese, have rates comparable to those of the other groups among women up to 45 yr old but have considerably lower rates in the older age groups. Population comparisons have two limitations: (a) even if a highly consistent relationship between estriol ratios and breast cancer risk is found, it will be difficult to rule out the possibility that the association is non-causal; and (b) breast cancer rates are changing rapidly in some parts of the world and there is evidence that a woman's risk is largely a function of the birth-cohort to which she belongs. These limitations suggest that, if the hypothesis is not discredited by the population comparisons, a prospective cohort study will eventually be needed.

2553 IDENTIFICATION OF (23S)-5β-CHOLESTANE-3-α,7α,12α,23,25-PENTOL IN CEREBROTENDINOUS XANTHOMATOSIS. (Eng.) Hoshita, T. (Hiroshima Univ.

Sch. Medicine, Kasumi, 1-2-3, Hiroshima, Japan); Yasuhara, M.; Kihira, K.; Kuramoto, T. *Steroids* 27(5): 657-664; 1976.

In order to identify a bile alcohol isolated from patients with a cerebrotendinous xanthomatosis, the synthesis of (23R)- and (23S)-β-cholestane-3α,7α,12α,23-25-pentols was undertaken. Norcholyl aldehyde was converted into the cholestanepentols by a Reformatsky reaction with ethyl bromoacetate, followed by a Grignard reaction with methylmagnesium iodide. The 23S epimer thus synthesized was identical with the bile alcohol in question. The bile alcohol resulted from an impaired capacity to convert cholesterol to C_{24} bile acids, resulting in the excretion of considerable amounts of C_{27} bile alcohols.

2554 DETECTION OF CARCINOGEN INDUCED DNA DAMAGE BY ALKALINE ELUTION (Proceedings Abstract). (Eng.) Petzold, G. (Upjohn Co., Kalamazoo, MI 49001); Harbach, P.; Swenberg, J. *Proc Am Assoc Cancer Res* 17: 31; 1976.

2555 THE MODULATION OF MEMBRANE DRUG PERMEABILITY (Proceedings Abstract). (Eng.) Carlsen, S. A. (Dept. Medical Biophysics, Univ. Toronto, Ontario, Canada M4X 1K9); Till, J. E. *Proc Am Assoc Cancer Res* 17: 34; 1976.

2556 MOUSE SERUM ESTERASES AS EARLY INDICATORS OF CHEMICAL CARCINOGEN ACTION (Proceedings Abstract). (Eng.) Tyndall, R. L. (ORNL and Oak Ridge Assoc. Univ., Oak Ridge, TN 37830); Clapp, N. K.; Burtis, C. A.; Andrews, G. *Proc Am Assoc Cancer Res* 17: 169; 1976.

2557 CHEMICAL IDENTIFICATION OF A CARCINOGEN PRESENT IN BEEF AND OTHER MEATS (Proceedings Abstract). (Eng.) Shamberger, R. J. (Cleveland Clinic, Cleveland, OH 44106); Willis, C. E. *Proc Am Assoc Cancer Res* 17: 222; 1976.

2558 EFFECTS OF MONOALKYLATING CARCINOGENS AND MUTAGENS ON TRANSFORMING DNA (Proceedings Abstract). (Eng.) Kubinski, H. (Univ. Wisconsin, Madison, WI 53706); Kubinski, Z. O. *Fed Proc* 35(7): 1660; 1976.

2559 MACROMOLECULAR ADDUCTS OF AFLATOXINS B1 AND B2 IN RAT LIVER IN VIVO (Proceedings Abstract). (Eng.) Lin, J.-K. (McArdle Lab., Univ. Wisconsin, Madison, WI 53706); Swenson, D. H.; Miller, E. C.; Miller, J. A. *Proc Am Assoc Cancer Res* 17: 38; 1976.

2560 HELICAL POLYSOMES INDUCED BY AFLATOXIN B_1 IN VIVO: A NEW HYPOTHESIS FOR HELIX FORMATION BY CHEMICALS AND CARCINOGENS. (Eng.) Sarasin, A. (Institut de Recherches Scientifiques sur le Cancer, 94800-Villejuif, France); Moule, Y. *Exp Cell Res* 97(2): 346-358; 1976.

2561 THE ANEMIA CAUSED BY AFLATOXIN. (Eng.) Tung, H.-T. (Dept. Poultry Science, North Carolina State Univ., Raleigh, NC 27607); Cook, F. W.; Wyatt, R. D.; Hamilton, P. B. *Poult Sci* 54(6): 1962-1969; 1975.

2562 POTENTIATION OF THE TOXIC ACTION OF AFLATOXIN B_1 BY PHENOBARBITAL (Proceedings Abstract). (Fre.) Moule, Y. (Inst. Recherches Scientifiques sur le Cancer, F-94800 Villejuif, France). *J Physiol (Paris)* 71(2): 302A; 1975.

2563 AFLATOXIN B_1 METABOLISM TO AFLATOXICOL AND DERIVATIVES LETHAL TO *BACILLUS SUBTILIS* GSY 1057 BY RAINBOW TROUT (*SALMO GAIRDNERI*) LIVER. (Eng.) Schoenhard, G. L. (Dept. Drug Metabolism, Searle Labs., Box 5110, Chicago, IL 60680); Lee, D. J.; Howell, S. E.; Pawlowski, N. E.; Libbey, L. M.; Sinnhuber, R. O. *Cancer Res* 36(6): 2040-2045; 1976.

2564 A FUNCTIONAL HETEROTOPIC BLADDER TRANSPLANTED TO THE BACK OF AN ISOLOGOUS RAT (Proceedings Abstract). (Eng.) Oyasu, R. (Northwestern Univ. Medical Sch., Chicago, IL 60611); Manning, D. J.; Matsumoto, M. *Proc Am Assoc Cancer Res* 17: 210; 1976.

2565 MULTIPLE SITES OF ACTION OF N-OH-2-ACETYLAMINOFLUORENE (N-OH-AAF) IN RAT HEPATIC NUCLEAR TRANSCRIPTION (Proceedings Abstract). (Eng.) Yu, F.-L. (Dept. Biochemistry, Thomas Jefferson Univ., Philadelphia, PA 19107). *Proc Am Assoc Cancer Res* 17: 63; 1976.

2566 BINDING OF N-HYDROXYACETYLAMINOFLUORENE TO RAT LIVER EU-AND HETEROCHROMATIN (Proceedings Abstract). (Eng.) Moyer, G. H. (Univ. California, Los Angeles, CA 90024); Austin, G. E. *Proc Am Assoc Cancer Res* 17: 177; 1976.

2567 N-ACETYLTRANSFERASE PHENOTYPE OF PATIENTS WITH ''SPONTANEOUS'' URINARY BLADDER CANCER (Proceedings Abstract). (Eng.) Lower, G. M., Jr. (Dept. Human Oncology, Univ. Wisconsin Medical Sch., Madison, WI 53706); Nilsson, T.; Bryan, G. T. *Proc Am Assoc Cancer Res* 17: 203; 1976.

2568 BINDING OF PYRIDOXAL 5'-PHOSPHATE TO TRNA ISOLATED FROM A HUMAN CELL LINE OF COLON CARCINOMA: MULTIPLE EFFECTS ON TRANSLATION (Proceedings Abstract). (Eng.) Kopelovich, L. (Memorial Sloan-Kettering Cancer Center, New York, NY 10021); Wolfe, G.; Lipkin, M. *Proc Am Assoc Cancer Res* 17: 142; 1976.

2569 THE MUTAGENIC ACTIVITY OF BIS-(2-AMINO-1-NAPHTHYL) PHOSPHATE AND SOME IN VITRO ACTIVATION PRODUCTS IN *SACCHAROMYCES CEREVISIAE* (Proceedings Abstract). (Eng.) Reinhold, R. W. (Dept. Environmental Medicine, New York Univ. Medical Center, New York, NY 10016); Troll, W.; Finkelstein, T. T.; Mukai, F. *Proc Am Assoc Cancer Res* 17: 212; 1976.

2570 ENHANCEMENT OF N-(4-(5-NITRO-2-FURYL)-2-THIAZOLYL)FORMAMIDE (FANFT) CARCINOGENICITY FOR RAT URINARY BLADDER BY ALLOPURINOL (Proceedings Abstract). (Eng.) Wang, C. Y. (Dept. Human Oncology, Univ. Wisconsin Center for Health Sciences, Madison, WI 53706); Hayashida, S. *Proc Am Assoc Cancer Res* 17: 149; 1976.

2571 ENHANCING EFFECT OF ALLOPURINOL ON THE INDUCTION OF BLADDER CANCER IN RATS BY N-[4-(5-NITRO-2-FURYL)-2-THIAZOLYL]FORMAMIDE. (Eng.) Wang, C. Y. (Univ. Wisconsin Center for Health Sciences, Madison, WI 53706); Hayashida, S.; Pamukcu, A. M.; Bryan, G. T. *Cancer Res* 36(5): 1551-1555; 1976.

2572 HISTOGENESIS AND GROWTH PATTERN OF 1,2-DIMETHYLHYDRAZINE-INDUCED RAT COLON ADENOCARCINOMA. (Eng.) Maskens, A. P. (Service d'Anatomie Pathologique, Cliniques Univ. Saint Pierre, 3000 Louvain, Belgium). *Cancer Res* 36(5): 1585-1592; 1976.

2573 LONG TERM CHEMOTHERAPY AND/OR IMMUNOTHERAPY OF PRIMARY MURINE BLADDER CANCER (Proceedings Abstract). (Eng.) Soloway, M. S. (Univ. Tennessee Sch. Medicine, Memphis, TN 38104); Martino, C. *Proc Am Assoc Cancer Res* 17: 9; 1976.

2574 COLO-RECTAL TUMORS IN INBRED MICE TREATED WITH 1:2 DIMETHYLHYDRAZINE (Proceedings Abstract). (Eng.) Diwan, B. A. (Jackson Lab., Bar Harbor, ME 04609); Meier, H. *Proc Am Assoc Cancer Res* 17: 106; 1976.

2575 EFFECT OF AGE AND SEX ON COLON CARCINOGENESIS (Proceedings Abstract). (Eng.) Moon, R. C. (IIT Research Inst., Chicago, IL 60616); Schiff, L. J.; Fricks, C. M. *Proc Am Assoc Cancer Res* 17: 23; 1976.

2576 ALKYLATING AND MUTAGENIC METABOLITES OF HALOGENATED OLEFINS PRODUCED BY HUMAN AND ANIMAL TISSUES (Proceedings Abstract). (Eng.) Bartsch, H. (International Agency for Res. on Cancer, 69008 Lyon, France); Malaveille, C.; Barbin, A.; Planche, G.; Montesano, R. *Proc Am Assoc Cancer Res* 18: 17; 1976.

2577 CAFFEINE ENHANCEMENT OF THE CYTOTOXIC AND MUTAGENIC EFFECTS OF ULTRAVIOLET RADIATION IN XERODERMA PIGMENTOSUM VARIANT STRAINS OF HUMAN CELLS (Proceedings Abstract). (Eng.) Maher, V. M. (Michigan Cancer Foundation, Detroit, MI 48201); Ouellette, L. M.; Curren, R. D.; McCormick, J. J. *Proc Am Assoc Cancer Res* 17: 138; 1976.

2578 ALTERATION IN THE FIDELITY OF DNA SYNTHESIS BY BERYLLIUM (Proceedings Abstract). (Eng.) Sirover, M. A. (Inst. for Cancer Res., Fox Chase Cancer Center, Philadelphia, PA 19111); Loeb, L. A. *Proc Am Assoc Cancer Res* 17: 113; 1976.

2579 HISTOLOGICAL STUDY OF IRON DEPOSITS IN MAMMARY GLANDS OF MICE (Proceedings Abstract). (Eng.) Seman, G. (Dept. Virology, Univ. Texas System Cancer Center, M. D. Anderson Hosp. and Tumor Inst., Houston, TX 77025); Bowen, J. M.; Dmochowski, L. *Proc Am Assoc Cancer Res* 17: 110; 1976.

2580 OXYGEN AND LIGHT EFFECTS ON CHROMOSOMAL ABERRATIONS IN MOUSE CELLS IN VITRO (Proceedings Abstract). (Eng.) Sanford, K. K. (Lab. Biochemistry, Natl. Cancer Inst., Bethesda, MD 20014); Parshad, R. *Proc Am Assoc Cancer Res* 17: 65; 1976.

2581 A NEW CHARACTERISTIC TRANSPLANTABLE TYPE OF BREAST CARCINOMA IN NB RATS FOLLOWING COMBINED ESTROGEN-ANDROGEN TREATMENT (Proceedings Abstract). (Eng.) Noble, R. L. (Cancer Res. Centre, Univ. British Columbia, Vancouver V6T 1W5). *Proc Am Assoc Cancer Res* 17: 221; 1976.

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2583 RHABDOMYOSARCOMA IN MICE MAINTAINED ON FAT ENRICHED DIETS (Proceedings Abstract). (Eng.) Szepsenwol, J. (Dept. Anatomy, Univ. Puerto Rico Sch. Medicine, San Juan, PR 00936). *Proc Am Assoc Cancer Res* 17: 217; 1976.

2584 A METABOLITE OF N-NITROSO-HEXAMETHYL-ENE-IMINE (NHI) BOUND TO RAT LIVER RNA (Proceedings Abstract). (Eng.) Ross, A. E. (Eppley Inst. for Res. in Cancer, Univ. Nebraska Medical Center, Omaha, NB 68105). *Proc Am Assoc Cancer Res* 17: 114; 1976.

2585 IN VITRO METHYLATION OF NUCLEIC ACID BY DIMETHYLNITROSAMINE AND ITS INHIBITION BY VARIOUS CHEMICAL INHIBITORS (Proceedings Abstract). (Eng.) Oshiro, Y. (NCI Frederick Cancer Res. Center, Frederick, MD 21701); Zielinski, W. L., Jr.; Harris, P. B. *Proc Am Assoc Cancer Res* 17: 104; 1976.

2586 CHARACTERIZATION OF DIMETHYLNITROSAMINE (DMN)-INDUCED METHYLATED PRODUCTS IN RAT LIVER DNA REPLICATED IN VIVO (Proceedings Abstract). (Eng.) Abanobi, S. E. (Fels Res. Inst., Temple Univ. Medical Sch., Philadelphia, PA 19140); Mulivor, R. A.; Rajalakshmi, S.; Sarmi, D. S. R. *Proc Am Assoc Cancer Res* 17: 103; 1976.

2587 CARCINOGENIC *N*-NITROSO-DIMETHYLAMINE FROM THE REACTION OF THE ANALGESIC AMIDOPYRINE AND NITRITE EXTRACTED FROM FOODSTUFFS. (Eng.) LeBar, J. (Medizinaluntersuchungsamt, D-4500 Osnabrueck, W. Germany); Sander J. *Z Krebsforsch* 84(3): 299-310; 1975.

2588 INFLUENCE OF DISULFIRAM ON THE ORGANOTROPY OF THE CARCINOGENIC EFFECT OF DIMETHYLNITROSAMINE AND DIETHYLNITROSAMINE IN RATS. (Eng.) Schmaehl, D. (Inst. f. Toxikologie u. Chemotherapie, Im Neuenhaimer Feld 280, D-6900 Heidelberg, W. Germany); Kreuger, F. W.; Habs, M.; Diehl, B. *Z Krebsforsch Klin Onkol* 85(3): 271-276; 1976.

2589 EXPERIMENTAL INDUCTION OF COLONIC CARCINOMAS IN RATS: ANALYSIS OF FACTORS INFLUENCING UPON THE INCIDENCE. (Eng.) Kanazawa, K. (Tokyo Metropolitan Geriatric Hosp., Yoikuin, Sakaecho, Itabashi-Ku, Tokyo 173, Japan); Yamamoto, T.; Sato, S. *Jpn J Exp Med* 45(6): 439-456; 1975.

2590 IN VITRO MODULATION OF MNNG-INDUCED NEOPLASTIC TRANSFORMATION BY CYCLIC NUCLEOTIDES (Proceedings Abstract). (Eng.) Mironescu, S. (Blood Res. Lab., American Natl. Red Cross, Bethesda, MD 20014); DiPaolo, J. A. *Proc Am Assoc Cancer Res* 17: 195; 1976.

2591 BCNU INDUCED DNA STRAND BREAKS AND EFFECTS ON GROWTH IN CULTURED NORMAL AND TRANSFORMED HUMAN CELLS (Proceedings Abstract). (Eng.) Erickson, L. C. (Div. Cancer Treatment, Natl. Cancer Inst., Natl. Insts. Health, Bethesda, MD 20014); Bradley, M. O. *Proc Am Assoc Cancer Res* 17: 134; 1976.

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2611 MORPHOLOGY, GROWTH AND ESTROGEN BINDING CAPACITY IN DMBA-INDUCED MAMMARY TUMORS (Proceedings Abstract). (Eng.) Boylan, E. S. (Queens Coll. CUNY, Flushing, NY 11367); Fowler, E. H.; Wittliff, J. L. *Proc Am Assoc Cancer Res* 17: 197; 1976.

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2616 RIBOSOME ACCUMULATION IN MOUSE INTERFOLLICULAR EPIDERMIS AND EPIDERMIS OF PAPILLOMAS DURING PROMOTION WITH TETRADECANOYL-PHORBOL-ACETATE (TPA) (Proceedings Abstract). (Eng.) De Young, L. M. (Syracuse Univ., Syracuse, NY 13210); Argyris, T. S.; Gordon, G. B. *Proc Am Assoc Cancer Res* 17: 20; 1976.

2617 EFFECT OF DMBA ON RAT MAMMARY GLAND N-ACETYLTRANSFERASES (Proceedings Abstract). (Eng.) Libby, P. R. (Roswell Park Memorial Inst., Buffalo, NY 14263). *Proc Am Assoc Cancer Res* 17: 187; 1976.

2618 *E. COLI* ENDOTOXIN EFFECT ON A METHYLCHOLANTHRENE INDUCED SARCOMA GROWN IN THE HAMSTER CHEEKPOUCH (Proceedings Abstract). (Eng.) Stevens, D. (Univ. Vermont, Burlington, VT 05401); Stinebring, W. *Proc Am Assoc Cancer Res* 17: 18; 1976.

2619 MATURATION OF UNDIFFERENTIATED EPITHELIUM OF RAT BASAL CELL CARCINOMA TRANSPLANTED INTO ESTRADIOL TREATED UTERUS (Proceedings Abstract). (Eng.) Cooper, M. (VA Hosp., Allen Park, MI); Pinkus, H. *Proc Am Assoc Cancer Res* 17: 37; 1976.

2620 EVALUATION OF IN VITRO TESTS FOR MALIGNANCY OF HAMSTER CELLS TRANSFORMED BY CHEMICAL (Proceedings Abstract). (Eng.) Hatch, G. (Biolabs, Inc., Northbrook, IL 60062); Casto, B.; Balwierz, P.; Goodheart, C. *Proc Am Assoc Cancer Res* 17: 22; 1976.

See also:

* (Rev): 2401, 2402, 2403, 2404, 2405, 2406, 2407, 2408, 2409, 2410, _411, 2412, 2434, 2438, 2453, 2458, 2469, 2470, 2471, 2472, 2473, 2474, 2475, 2476, 2477, 2478, 2500, 2502, 2509
* (Phys): 2625, 2626
* (Viral): 2664
* (Immun): 2757, 2770, 2772, 2774, 2776, 2778, 2780, 2786, 2789, 2790, 2810
* (Path): 2832, 2841, 2848, 2868, 2877, 2878, 2880, 2884, 2899, 2900, 2906, 2907, 2908, 2909
* (Epid-Biom): 2921, 2923, 2924, 2925, 2926, 2927, 2928, 2937, 2942, 2943, 2949

2621 RESISTANCE OF HUMAN MELANOMA CELLS TO ULTRAVIOLET RADIATION. (Eng.) Chalmers, A. H. (Dept. Biochemistry, Univ. Queensland, Brisbane 4067, Australia); Lavin, M.; Atisoontornkul, S.; Mansbridge, J,; Kidson, C. *Cancer Res* 36(6): 1930-1934; 1976.

A series of five human melanoma cell lines (MM96, MM127, MM138, MM170, and MM200) was shown to be a highly resistant to ultraviolet (UV) radiation, with a D_0 of 400 ergs/sq mm. Melanotic melanoma cells increased their production of melanin following UV radiation, whereas some amelanotic cells did not. Melanotic and amelanotic melanoma cell lines exhibited the same UV resistance; melanoma and nonmelanoma cells formed the same numbers of thymine dimers at a given UV dose. These data imply that melanin does not play a major role in protecting the DNA of melanoma cells against UV damage in culture. The rates of removal of thymine dimers from the DNA of melanoma cells were comparable to those in UV-sensitive, nonmelanoma cell lines, so that rapid excision repair does not explain UV resistance in the melanoma cells. No DNA strand breakage was detected in a melanoma cell line at moderate UV doses.

2622 DNA-PROTEIN CROSS-LINKING BY ULTRAVIOLET RADIATION IN NORMAL HUMAN AND XERODERMA PIGMENTOSUM FIBROBLASTS. (Eng.) Fornace, A. J., Jr. (Dept. Pathology, 721 Huntington Ave., Boston, MA 02115); Kohn, K. W. *Biochim Biophys Acta* 435(1): 95-103; 1976.

DNA-protein cross-linking by UV radiation was measured in human fibroblasts by an adaptation of the DNA alkaline elution method. To measure cross-linking, a controlled frequency of DNA single-strand breaks was introduced by exposing the cells to a low dose of x-ray at 0 C prior to analysis by alkaline elution. The effect of prior exposure to UV radiation was to reduce the rate and/or extent of DNA elution from the x-irradiated cells. This effect was attributed to DNA-protein cross-linking, since the effect was reversed by treatment of the cell lysates with proteinase-k. Cross-linking in normal human fibroblasts occurred immediately after UV irradiation, prior to the appearance of DNA single-strand breaks due to excision repair. Upon incubation of normal cells after exposure to the radiation, the cross-linking was partially repaired. In xeroderma pigmentosum cells, cross-links appeared as in normal cells, but there was no repair. Instead, the extent of cross-linking appeared to increase upon incubation after irradiation. The reason for this increase in cross-linking is not known, but it could be a secondary damage reflecting severe cell damage, and the persistence of these links could prove to be more damaging than the UV radiation.

2623 IN VITRO REPAIR OF RADIATION-INDUCED STRAND BREAKS IN DNA. (Eng.) Mitzel-Lanbeck, L. (Institut fuer Strahlenbiologie, Kernforschungszentrum Karlsruhe, D 75 Karlsruhe 1, Postfach 3640 W. Germany); Schutz, G.; Hagen, U. *Biochim Biophys Acta* 432(2): 145-153; 1976.

DNA, γ-irradiated in solutions (400 μg/ml) or in isolated thymocytes, was treated with several enzymes to achieve rapair of the radiation-induced single strand breaks. Incubation with polynucleotide ligase joined only 25% of the single strand breaks; combined treatment with exonuclease III, DNA polymerase I, and polynucleotide ligase led to repair of 80% of the breaks. Tests for successful rejoining of the strand breaks were performed by showing the loss of primer 3'-OH sites of DNA polymerase I, by the resistance of incorporated nucleotides in the gap to removal by a second exonuclease III treatment, and by strand break determination in the analytical ultracentrifuge. Twenty percent of the radiation-induced strand breaks were not repaired by this combined treatment, possibly due to an incomplete binding of the ligase on the 5'-terminals and/or an incomplete removal of the damaged 3'-terminals by exonuclease III. Removal of damaged end groups on the 3'-terminal may be an essential step in the repair of strand breaks.

2624 REPAIR OF ULTRAVIOLET-DAMAGED DNA IN VERTEBRATE CELLS. (Eng.) Yatani, R. (Mie Univ. Sch. Medicine, Tsu, Japan); Togo, Y.; Maegawa, K.; Aoki, N.; Aketa, S.; Takanari, H. *Mie Med J* 25(1): 65-80; 1975.

The distribution of the three repair systems for UV-damaged DNA in vertebrates, photoreactivation, caffeine-sensitive repair, and excision repair, was studied by chromatography, autoradiography, and caffeine inhibition on survival fractions of a few permanent and primary culture cell lines of fish, amphibians, reptiles, fowl, and mammals. The results suggest that cells from the Pisces, Amphibia, and Reptilia have the most active photoreactivation. The cells from the Rodentia and from the Aves had caffeine-sensitive but weak excision repair. Monkey cells also have both caffeine-sensitive repair and weak excision repair, but human cells have only excision repair, with the highest activity in the vertebrate cells examined. Chimpanzee cells show a transitional type from the monkey pattern to the human pattern: these cells had active excision repair, combined with caffeine-sensitive repair.

2625 EFFECT OF DNA REPAIR ON THE CYTOTOXICITY AND MUTAGENICITY OF UV IRRADIATION AND OF CHEMICAL CARCINOGENS IN NORMAL AND XERODERMA PIGMENTOSUM CELLS. (Eng.) Maher, V. M.; McCormick, J. J. In: *Biology of Radiation Carcinogenesis*. Edited by J. M. Yuhas, R. W. Tennant, and J. D. Regan. (New York: Raven Press): pp. 129-145; 1976.

The cytotoxic and mutagenic action of UV irradiation and of aromatic amides or polycyclic hydrocarbons was compared quantitatively in normally repairing strains of human cells and in several excision-repair deficient or postreplication repair-deficient xeroderma pigmentosum (XP) strains. The survival of the cloning capacity of the various excision-defective XP strains following exposure to UV or the carcinogens reflected the excision capacity of the particular strain tested. Cells from XP variants showed lower survival than normal cells and exhibited a markedly reduced capacity for postreplication repair (gap-filling) of UV lesions. An excision-defective XP strain from complementation groups A and C was compared with normal cells for the frequency of mutations induced by various doses of UV. The XP strains showed an increased frequency, which correlated with their sensitivity to UV and their capacity for excision repair of UV-induced lesions. The induced mutation frequencies of the two XP strains and the normal strain, examined as a function of the percent survival (cytotoxicity) of the particular strain, were equal. This is evidence of an error-free excision repair process in human cells. The frequency of mutations induced in normal cells and in the group C XP strain by active aromatic carcinogens was compared. For each compound, the XP strain exhibited a significantly higher mutation frequency than that of the normal. These mutation frequencies, examined as a function of percent survival of the strains, were equal. It is hypothesized that the three strains, although receiving very different UV doses, contain the same average of unrepaired lesions at the time of some critical event because of their different rates of DNA excision repair; these unrepaired lesions are responsible for both the mutagenic and lethal effects of the irradiation.

2626 THE MOLECULAR MECHANISM OF INDUCED MUTATIONS AND AN IN VITRO BIOCHEMICAL ASSAY FOR MUTAGENESIS. (Eng.) Radman, M.; Caillet-Fauquet, P.; Defais, M.; Villani, G. In: *Screening Tests in Chemical Carcinogenesis. Meeting Held in Brussels, 9-12 June 1975.* International Agency for Res. on Cancer. (Brussels, Belgium): no. 12, pp. 537-548; 1976.

Mutation of *Escherichia coli* DNA by either chemical mutagens or irradiation results in the induction of a new DNA repair system, called SOS repair. This system enables DNA replication to continue, but the replication process becomes very error-prone. Mutants deficient in SOS repair are rec A- and lex A-. Carcinogens/mutagens may induce a similar SOS error prone system in mammalian cells, and an assay procedure for the detection of any such activity is described. The assay measures the misincorporation of deoxyguanidine and deoxycytidine triphosphates into a poly dT:oligo dA template-primer that has been UV-irradiated to produce thymine dimers. (16 references)

2627 THYROID CANCER OCCURRING AS A LATE CONSEQUENCE OF HEAD-AND-NECK IRRADIATION. EVALUATION OF 1056 PATIENTS. (Eng.) Favus, M. J. (Michael Reese Hosp. and Medical Center, 2900 S. Ellis Ave., Chicago, IL 60616); Schneider, A. B.; Stachura, M. E.; Arnold, J. E.; Ryo, U. Y.; Pinsky, S. M.; Colman, M.; Arnold, M. J.; Frohman, L. A. *N Engl J Med* 294(19): 1019-1025; 1976.

From January 1 to September 30, 1974, 1056 of 5266 subjects (20.1%) were examined who had received therapeutic irradiation, primarily for infections and inflammatory disease of the upper respiratory tract during the 1940's and 1950's. The tonsillar and nasopharyngeal region was the treatment site in 85% of those examined. Palpable nodular thyroid disease was found in 16.5%, and nonpalpable lesions were detected by ^{99}mTc pertechnetate thyroid imaging in an additional 10.7%, for a prevalence of nodular disease of 27.2%. Operation on 71% with nodular disease revealed thyroid cancer in 33% (60 of 182). Preliminary analysis for potential risk factors suggested a correlation between radiation exposure and the presence of thyroid nodules. These findings indicate that nodular thyroid disease, both benign and malignant, continues as a major health problem for at least 35 yr in exposed subjects.

2628 DIAGNOSTIC AND THERAPEUTIC RADIATION EXPOSURE. (Eng.) Russell, W. J. (Radiation Effects Res. Foundation, Hiroshima, 730 Japan). *J Radiat Res* 16(Suppl.): 42-48; 1975.

Diagnostic and therapeutic radiology were studied as possible contaminants in the evaluations of A-bomb survivors in the Atomic Bomb Casualty Committee (ABCC)-Japanese National Institute of Health, Adult Health Study for radiation effects. Hiroshima and Nagasaki subjects received x-ray examinations elsewhere within 3 mo of their ABCC visits at rates of 23 and 12%, respectively. Medical x-ray examinations were more frequent among survivors than comparison subjects. Hiroshima and Nagasaki radiologic practice steadily increased since 1948 and differed markedly by city. From 1946 to 1970, the Hiroshima and Nagasaki x-ray bone marrow doses were 2,300 and 1,000 g-rads, respectively. By 1970, cumulated medical x-ray doses approximated A-bomb doses at distances of 2,000 m from Hiroshima and 2,800 m from Nagasaki. ABCC x-ray examination doses per subject were routinely updated for comparison with A-bomb doses. Each subject's reported fluoroscopy, photofluorography, and radiation therapy exposure elsewhere were recorded for future reference. Dental radiography, though increasing, was not an important contributor to survivors' overall exposure. Radiation therapy exposures of 137 subjects were confirmed, and doses were estimated for most. Two-thirds of the treatments were for malignancies, with therapy differing markedly by city. Five cancers possibly arose from earlier radiation therapy. The importance of considering diagnostic and therapeutic radiology when attributing diseases to the atomic bombs is underscored by the results.

2629 IDENTIFICATION OF PARTICLES IN OVARIAN TISSUE. (Eng.) Henderson, W. J.; Griffiths, K. In: *Gynaecological Malignancy: Clinical and Experimental Studies.* Edited by M. G. Brush; R. W. Taylor. (Baltimore: Williams and Wilkins): pp. 225-240; 1975.

Electron microscopic examination of polyvinyl alcohol replicas of normal and malignant ovarian tissue revealed the presence of crystalline talc, some crystals being observed within the cells. The particles were verified as having the expected 3:1 silicon:magnesium ratio of talc by analysis of the energy spectrum of x-rays emitted by in situ bombardment with electrons. Incineration of wet tissue at 500 C in oxygen was effective in producing a sample of particulate material for the analysis of individual crystals. The transfer in water of the ashed material from a known weight of ovarian tissue to numbered reference grids and subsequent drying in air provided a means of quantitation. This procedure indicated that there were between 2.5 and 40 x 10^5 particles of talc per gram of wet wt normal ovarian tissue. Other particulate material such as kaolin particles and diatoms were also found. The possibility that embedded talc crystals may be responsible for early carcinomatous changes is worthy of further consideration. (21 references)

2630 RADIATION CARCINOGENESIS-LABORATORY STUDIES. (Eng.) Shellabarger, C. J. (Upton, NY: Brookhaven Natl. Lab.): 28 pp.; 1975.

2631 FAST-NEUTRON IRRADIATION OF MOUSE EMBRYOS IN THE PRONUCLEAR ZYGOTE STAGE: MORTALITY CURVES AND NEOPLASTIC DISEASES IN 30-DAY POSTNATAL SURVIVORS (39312). (Eng.) Friedberg, W. (Civil Aeromedical Inst., Oklahoma City, OK 73125); Hanneman, G. D.; Faulkner, D. N.; Neas, B. R.; Cosgrove, G. E., Jr.; Darden, E. B., Jr. *Proc Soc Exp Biol Med* 151(4): 808-810; 1976.

2632 ALPHA-EMITTERS (Letter to Editor). (Eng.) Wiley, J. S. (Saratoga, CA). *Am Sci* 63(6): 619; 1975.

2633 METABOLIC AND DOSIMETRIC STUDIES AFTER INHALATION OF ^{227}TH IN RATS WITH REGARD TO THE RISK OF LUNG AND BONE TUMOURS. (Eng.) Mueller, W. A. (Institut fuer Biologie der Gesellschaft fuer Strahlen- u. Umweltforschung mbH, D-8042 Neuherberg, Ingolstadter Landstr. 1, W. Germany); Nenot, J. C.; Daburon, M. L.; Lafuma, J. *Radiat Environ Biophys* 11(4): 309-318; 1975.

2634 DULUTH WATER REVISED (Letter to Editor). (Eng.) Coleman, J. R. (Minneapolis, MN). *Minn Med* 59(2): 95-97; 1976.

See also:

* (Rev): 2413, 2414, 2415, 2416, 2460, 2479, 2480
* (Chem): 2577, 2580
* (Viral): 2651
* (Immun): 2769, 2782
* (Path): 2830, 2882, 2917
* (Epid-Biom): 2938, 2947

2635 UNINTEGRATED VIRAL DNA IS SYNTHESIZED IN THE CYTOPLASM OF AVIAN SARCOMA VIRUS-TRANSFORMED DUCK CELLS BY VIRAL DNA POLYMERASE. (Eng.) Varmus, H. E. (Dept. Microbiology, Univ. California, San Francisco, CA 94143); Shank, P. R. *J Virol* 18(2): 567-573; 1976.

The location, structure, and mechanism of synthesis of unintegrated viral DNA present in fully transformed cultures of avian sarcoma virus-infected duck cells were examined. De novo synthesis of the unintegrated forms several weeks after the initial infection was documented by labeling unintegrated DNA in both strands with 5-bromodeoxyuridine. The unintegrated DNA was synthesized in, and probably confined to, the cytoplasm; it consisted of duplexes of short ''plus'' strands (about 0.5 x 10^6 to 1.0 x 10^6 daltons) and ''minus'' strands the length of a subunit of the viral genome (about 2.5 x 10^6 to 3.0 x 10^6 daltons). The structure of the duplex and the mode of incorporation of the density label support the hypothesis that the unintegrated DNA is synthesized from an RNA template by virus-coded DNA polymerase.

2636 EVIDENCE FOR METHYLATION OF B77 AVIAN SARCOMA VIRUS GENOME RNA SUBUNITS. (Eng.) Stoltzfus, C. M. (Vanderbilt Univ. Sch. Medicine, Nashville, TN 37232); Dimock, K. *J Virol* 18(2): 586-595; 1976.

B77 avian sarcoma virus RNA was labeled with [methyl-^{3}H]methionine under conditions that prevented nonmethyl incorporation of ^{3}H radioactivity into purine rings and studied by chromatography and electrophoresis. As seen from the determined values for the extent of methylation of 4S RNA isolated from infected chicken embryo cells, approximately 30S to 40S RNA subunits that resulted from heat denaturation of the 60S to 70S RNA contained about 21 methyl groups (of which 14 to 16 were present at internal positions as N^6-methyladenosine residues). Each of the virion RNA subunits also appeared to contain about two methyl groups in the ''capped'' 5'-terminal structure m^7-G(5')ppp(5')Gm. These results support the hypothesis that the 30S to 40S genome RNA of oncornaviruses also serves an mRNA function in infected cells.

2637 USE OF ALKALINE SUCROSE GRADIENTS IN A ZONAL ROTOR TO DETECT INTEGRATED AND UNINTEGRATED AVIAN SARCOMA VIRUS-SPECIFIC DNA IN CELLS. (Eng.) Varmus, H. E. (Dept. Microbiology, Univ. California, San Francisco, CA 94143); Heasley, S.; Linn, J.; Wheeler, K. *J Virol* 18(2): 574-585; 1976.

Integrated and unintegrated forms of avian sarcoma virus-specific DNA in cells were distinguished by sedimentation through an alkaline sucrose gradient in a slowly reorienting zonal rotor. The cell types were chick cells that inherit viral genes, virally transformed but non-permissive mammalian cells, transformed virus-producing avian cells, and acutely infected avian cells. Most, if not all, viral DNA appeared completely integrated into the host cell genome in an avian sarcoma virus-transformed mammalian cell and in normal chicken cells (in which viral DNA is genetically transmitted). Fully transformed duck cells and duck embryo fibroblasts infected for 20 to 72 hr contained both integrated and unintegrated viral DNA; up to one copy per cell was integrated within 20 hr after infection, and four to eight copies were integrated in fully transformed cells. The amount of unintegrated DNA varied but it comprised over 75% of the viral DNA in acutely infected cells and from 20% to 70% of the viral DNA in fully transformed cells. The unintegrated DNA in either case consisted principally of duplexes with ''minus'' strands the length of a subunit of the viral genome (2.5 x 10^6 to 3 x 10^6 daltons) and relatively short ''plus'' strands (0.5 x 10^6 to 1.0 to 10^6 daltons).

2638 THE PRESENCE OF COMPLEMENT FIXING ANTIBODIES TO ROUS SARCOMA VIRUS (SCHMIDT-RUPPIN STRAIN) IN PATIENTS WITH VARIOUS FORMS OF NEOPLASIA. (Eng.) Nastac, E. (The ''Stefan S. Nicolau'' Inst. Virology, 285, Sos. Mihai Bravu, Bucharest, Romania); Stoian, M.; Lungu, M.; Ionescu, T.; Athanasiu, P. *Rev Roum Med Virol* 26(1): 47-50; 1975.

The sera of patients with neoplasia or acute or chronic nonneoplastic diseases, of normal blood donors, and of fowl and cattle farm workers were studied for the presence of Schmidt-Ruppin strain Rous sarcoma virus antibodies. Antibodies were detected by complement fixation. One of 30 cattle farm workers tested positive (3.3%), as did 16 of 128 normal donors (12%), 13 of 67 acute leukemia patients (19.7%), 16 of 119 chronic leukemia patients (13.4%), 31 of 160 system disease patients (19.3%), 70 of 203 tumor patients (34.4%), 11 of 89 acute and chronic nonneoplastic disease patients (12.3%), and 7 of 33 fowl farm workers (21.2%). It is concluded that high percentages of complement fixing antibodies to this strain of Rous sarcoma virus occur in man, particularly in patients with various organ tumors.

2639 INFECTIVITY OF ROUS SARCOMA CELL DNA: COMPARISON OF TWO TECHNIQUES OF TRANSFECTION ASSAY. (Eng.) Hillova, J. (Inst. Cancerology and Immunogenetics, CNRS, 14, Avenue Paul-Vaillant-Couturier, F-94800 Villejuif, France); Hill, M.; Goubin, G.; Dantchev, D. *Intervirology* 5(6): 367-374; 1975.

A DNA extracted from a clone of chicken cells transformed by the Schmidt-Ruppin strain of Rous sarcoma virus, subgroup D, was assayed for infectivity by the calcium technique, which was then compared to the DEAE (diethylaminoethyl) dextran technique. Both procedures gave rise to viruses in transfection assays with both native and denatured (S1 nuclease susceptible) DNA's. The efficiency of these transfection techniques with native DNA was compared and found to be about the same, provided that with the calcium technique carrier DNA was used to complement DNA concentrations lower than 2.5 μg/ml.

2640 MORPHOLOGICAL CHANGES IN CHICK EMBRYO CELL CULTURES INDUCED BY AVIAN LEUKOSIS VIRUSES. (Eng.) Oker-Blom, N. (Dept. Virology, Univ. Helsinki, Haartmaninkatu 3, SF-00290 Helsinki 29, Finland); Kallio, A.; Hortling, L. *Intervirology* 5(6): 342-353; 1975.

Morphological alterations, which may be significant in the oncogenicity of the leukosis viruses, were observed after 5-15 serial passages of primary chick embryo cells infected with three different strains of avian leukosis virus (OK 10, an A subgroup virus isolated from a natural infection; RAV-1, an established laboratory strain of the A subgroup; and RAV-2, a laboratory strain of the B subgroup). The infected cells had a prolonged lifespan of approximately 28 passages, compared to 14 passages for control cells. Since the altered cells had none of the attributes of transformed cells, such as growth in soft agar, loss of contact inhibition, tumor formation in chickens, or loss of fibroblast surface antigen, the changes are considered conversions rather than transformations. The morphological changes differed depending upon the subgroup of the inducing virus. Cells converted with the A subgroup viruses were uniformly epithelioid, whereas cells converted with the B subgroup virus were less uniform in size and shape. The occurrence of these altered cells could be the result of acquisition of new properties and/or disappearance of competing cells. The cells in converted cultures could either be fibroblasts with a changed morphology or nonfibroblastic cells that normally do not multiply in the presence of fibroblasts or that gain a better growth potential when infected. The presence of the fibroblast surface antigen would support the first possibility. The fact that the ''nontransforming'' avian leukosis viruses selected given types of in vitro cells implies that a similar event occurs in vivo and that leukosis actually results from a combined effect of conversion and transformation.

2641 INITIATION OF DNA SYNTHESIS BY THE AVIAN ONCORNAVIRUS RNA-DIRECTED DNA POLYMERASE: STRUCTURAL AND FUNCTIONAL LOCALIZATION OF THE MAJOR SPECIES OF PRIMER RNA ON THE ONCORNAVIRUS GENOME. (Eng.) Staskus, K. (Univ. Minnesota Medical Sch., Minneapolis, MN 55455); Collett, M. S.; Faras, A. J. *Virology* 71(1): 162-168; 1976.

The structural and functional localization of the binding site of the major species of primer RNA on the avian myeloblastosis virus (AMV) RNA genome was studied. Analysis of the saturation kinetics of the template activity reconstitution of 35S RNA with varying concentrations of purified primer RNA indicated the presence of one primer site per 35S RNA subunit. Functional localization was accomplished by reannealing purified primer RNA to various size-class fragments of the AMV genome to try to determine the minimum size-class fragments containing the primer site and therefore capable of actively participating as the template primer for DNA synthesis by RNA-directed DNA polymerase. Poly(A) sequences at the 3' terminus of AMV RNA were used to localize the primer site with respect to the 3' end of the genome. Of the various poly(A) fragments with a size range between 200 nucleotides and genome length, only the largest (β 35S) were capable of reannealing to primer RNA and becoming an active template·primer complex for AMV DNA polymerase. The results indicate that the primary site of binding for primer RNA is at or near the 5' end of the AMV RNA genome. Polyacrylamide gel electrophoresis of the poly(A) fragments subsequent to reannealment indicated that the site is located within 10% of the 5' terminus. The implications of these findings on the transcription of the oncornavirus genome in vitro and in vivo are discussed.

2642 *E. COLI* tRNAs AS INHIBITORS OF VIRAL REVERSE TRANSCRIPTION IN VITRO. (Eng.) Cavalieri, L. (Sloan-Kettering Inst. Cancer Res., 145 Boston Post Rd., Rye, NY 10580); Yamaura, I. *Nucleic Acids Res* 2(12): 2315-2328; 1975.

Addition of tRNA's from *Escherichia coli* to 70S avian myeloblastosis virus (AMV) RNA and AMV reverse transcriptase resulted in a dose-dependent inhibition of reverse transcription. Approximately 50% inhibition was observed at a wt ratio (70S RNA: tRNA) of 1:0.5 over an enzyme concentration of 1-4 U/0.1 ml. Sucrose velocity density gradient centrifugation revealed that tRNA was bound to the enzyme and not to 70S RNA. At low ionic strengths (0.06-0.08) the inhibitory effect of *E. coli* tRNA's was negligible, suggesting that oligomerization results in the loss of binding sites for these tRNA's even though the oligomeric forms retain the tRNATrp primer site(s) for the 70S AMV RNA. The inhibitory effects of a number of nonvirion tRNA's is being studied in various in vivo systems.

2643 A TRANSOVARIAN TRANSMISSION STUDY OF MAREK'S DISEASE IN JAPANESE QUAIL. (Eng.) Khare, M. L. (Dept. Animal Sciences, Thompson Hall, Cook Coll. Rutgers Univ., New Brunswick, NJ 08903); Grun, J.; Adams, E. V. *Poult Sci* 54(6): 1953-1961; 1975.

To determine if transovarian transmission of the JM strain of Marek's disease virus (MDV) occurs in Japanese quail, parent quail were infected with MDV when 1 day old. In another experiment, parent quail were infected when 7 wk old. No precipitating antigen or cytopathic or infective agent was isolated from the egg yolks, embryos, newly hatched quail, or progeny quail of the infected parents. During the 8-wk observation period, the progeny of infected parents showed no clinical symptoms, mortality, gross or microscopic lesions, MD viremia, or antibody. However, an MD-specific fluorescent antigen was observed in fibroblast and kidney cell cultures from embryos of parents infected at 1 day of age. Approximately 10% of the embryos contained this fluorescent antigen. Fluorescent antigen was not observed in cultures from embryos of quail infected at the age of 7 wk or control quail. These results indicate that complete, infective MDV is not transmitted via eggs in quail. Detection of specific fluorescence in embryos from infected quail is possibly due to a transmission of the viral genome of this highly cell-associated virus. The fluorescent antigen might also be a product of viral origin, such as protein. The age of the parent at the time of infection might also have influenced the presence or absence of the antigen in embryos.

2644 NUCLEOTIDE SEQUENCE HOMOLOGIES AMONG MOUSE MAMMARY TUMOR VIRUSES. (Eng.) Ringold, G. M. (Dept. Microbiology, Univ. California, San Francisco, CA 94143); Blair, P. B.; Bishop, J. M.; Varmus, H. E. *Virology* 70(2): 550-553; 1976.

Competition hybridizations were performed to determine the nucleotide sequence homology among the genomes of several mouse mammary tumor viruses (MMTV). DNA was made complementary to the entire genome of MMTV produced by GR tumor cells (MMTV-P), and the ability of RNAs from other virus strains (MMTV-L and MMTV-S) to compete with labeled RNA from MMTV-P for annealing to this DNA was tested. RNA from these strains was found to compete with 96-100% of labeled RNA from MMTV-P, indicating that the genomes of MMTV viruses are composed of closely related nucleotide sequences.

2645 METHODS OF OBTAINING ANTISERA AGAINST ANTIGENS OF MAMMARY GLAND CARCINOMA VIRUS. (Eng.) Kryukova, I. N. (Acad. Medical Sciences USSR, Moscow, USSR); Miller, G. G.; Frolova, T. Y. *Bull Exp Biol Med (USSR)* 80(9): 1133-1136; 1975.

Various methods of obtaining virus-containing preparations from mouse mammary gland tumors for the preparation of rabbit antisera against antigens of mammary gland carcinoma virus (MGCV) were tested. The following methods were used: (a) immunization of rabbits or rats with mammary gland tissue homogenates from high cancer or noninbred pregnant or lactating mice, followed by absorption of the resulting sera with normal tissue homogenates and the sera and milk of mice of low cancer strains; (b) immunization of rabbits with fractions with a buoyant density of 1.16 g/cm^3, isolated from homogenates of mammary gland carcinoma and mammary glands of high cancer mouse strains in a sucrose density gradient; and (c) immunization of rabbits with MGCV precipitated from the culture medium of mammary gland carcinoma growing in vitro and purified in a density gradient of sucrose or urographin. Methods a and c were actually tested; the MGCV in preparations obtained by method b was tested with the sera obtained. The tissues used were mammary glands and spontaneous and transplanted mammary gland carcinomas from C3H/He and C3H/Sn mice. Of the two methods tested, method c was more effective. It led to definite production of antibodies against MGCV antigen in one of two rabbits. After contaminating antibodies against normal mouse tissue and serum antigens and bovine serum were removed, the antiserum gave a single clear precipitation line.

2646 STUDY OF THE COMPOSITION OF LIPIDS AND PROTEINS IN TWO STRUCTURES ISOLATED FROM THE MILK OF INFECTED MICE: MAMMARY TUMOR VIRUS AND THE MEMBRANE OF MILK GLOBULES. (Fre.) Calberg-Bacq, C.-M. (Laboratorie de Microbiologie generale et medicale, Universite de Liege, Liege, Belgium); Francois, C.; Gosselin, L.; Osterrieth, P. M.; Rentier-Delrue, F. *Arch Int Physiol Biochim* 83(5): 948-949; 1975.

The biochemical study of the envelope of the mammary tumor virus and of the membrane of fatty milk globules (FMG) of a mouse showed that phosphatidylethanolamine was the major component of both. Both had high levels of phosphatidylserine and sphingomyelin as well as elevated choline and non-choline ratios. The virus, however, had three times as many lipids and a higher cholesterol:phospholipid ratio than the FMG. The virus also had seven glycopolypeptides as opposed to four in FMG. If, as hypothesized, these two structures are connected with the plasma membrane of the secretory cells, then the differences between them suggest that their secretion through budding would be consecutive to a membranous reorganization into structurally specific localized units.

2647 ONCORNAVIRUS GENES AND HOST PROTEINS. (Eng.) Del Villano, B. C. (Cleveland Clinic, 9500 Euclid St., Cleveland, OH 44106); Nave, B.; Lerner, R. A. *Bibl Haematol (Basel)* (43): 110-114; 1976.

Evidence that the gp70 of murine leukemia viruses (MuLV) is structurally pleomorphic is presented. The apparent molecular wt of gp70 from bone marrow cells, thymus cells, and sperm cells from an NZB mouse were approximately 77,000, 70,000, and 67,000, respectively. Antigenic cross-reactivity between MuLV gp70 and the gp70 of the secretions of the epididymis was demonstrated by Ouchterlony double diffusion. Antibodies prepared against purified MuLV gp70 gave a reaction of partial identity; the MuLV gp70 has antigenic determinants not present on the epididymal protein, resulting in a spur in this Ouchterlony. Differences in the interspecies determinants were demonstrated by competition radioimmune assays. Analysis of tryptic peptide fingerprints demonstrated that there might be marked differences in the primary structure of immunologically related gp70 from various sources. Treatment of gp70 with pronase completely inactivated the antigenicity of gp70, thereby eliminating the possibility that the cross-reactivity was the result of carbohydrate antigens. (14 references)

2648 RELATIONSHIP BETWEEN THE ONCORNAVIRUS GENE PRODUCT GP70 AND A MAJOR PROTEIN SECRETION OF THE MOUSE GENITAL TRACT. (Eng.) Del Villano, B. C. (Scripps Clinic and Res. Foundation, La Jolla, CA 92037); Lerner, R. A. *Nature* 259(5543): 497-499; 1976.

The presence of a protein related immunologically to the murine leukemia virus (MuLV) gp70 in the secretions of the epididymis and ductus deferens of the mouse is reported. This protein had a molecular wt of 67,000 and was associated with the surface of sperm. Polyacrylamide gel electrophoresis of indirect immune precipitates and analysis of peptide maps indicated that anti-gp70 antibody reacted specifically with a single component from purified MuLV, surface radioiodinated MuLV-producing lymphoblastoid cells, and secretions from the epididymis and ductus deferens. Antigenic cross-reactivity between MuLV gp70 and the genital tract gp70 was confirmed by double diffusion studies. As shown by competition radioimmunoassay with the interspecies assay, the relative amount of gp70 in the genital tract secretions varied from 2-10%. The MuLV gp70 and the molecules from the genital tract were immunologically similar, but not identical. Differences in the peptide maps of the host and viral proteins strongly suggested that they were transcripts from different genes. The data indicate that several distinct genes from gp70 can be found in the mouse genome; some of these code for proteins made by certain differentiated cells, while others code for proteins that are incorporated into virus.

2649 NEW GENE LOCUS MODIFYING SUSCEPTIBILITY TO CERTAIN B-TROPIC MURINE LEUKEMIA VIRUSES. (Eng.) Decleve, A. (Stanford Univ. Sch. Medicine, Stanford, CA 94305); Niwa, O.; Kojola, J.; Kaplan, H. S. *Proc Natl Acad Sci USA* 73(2): 585-590; 1976.

In vivo and in vitro assays were made of a new gene, SRV (so-called because it was first discovered to modify to RadLV, radiation leukemia virus), the dominant allele of which occurs in strain C57BL mice. The assays indicated that it independently modified the slope or ''hitness'' and level of sensitivity of the titration curves obtained when one subclass of B-tropic murine leukemic viruses (RadLV) is propagated on nonpermissive Fv-1 genotypes of mouse embryo cells. Replication of another subclass of B-tropic viruses (Tennant type) is not modified with respect to hitness, and that of N-tropic viruses is not affected. The SRV gene might act by modifying the dual restriction mechanism in such a way that the host cells acquire new viral recognition capabilities; it does not act through the induction of infectious xenotropic viruses.

2650 THYMIC HORMONE MODULATION OF LEUKEMOGENIC VIRUS REPLICATION. (Eng.) Forger, J. M., III (Harvard Sch. Public Health, Boston, MA 02115); Cerny, J. *Cancer Res* 36(6): 2048-2052; 1976.

The effect of purified calf thymus extract, thymosin, on injection with murine leukemia virus (MuLV) (Rauscher) was studied in adult thymectomized BALB/c mice. The course of infection was determined by virus titer in the blood plasma, by enumeration of spleen cells replicating MuLV (infectious centers), and by the immunofluorescent assay of cells for expression of virus-induced cell membrane antigen. Thymectomy (performed 1 wk prior to the virus injection) decreased MuLV infection by all three parameters. Administration of thymosin (500 μg, given in equal doses, 2 days before and at the time of injection of virus) caused a marked increase of plasma titer of virus and an increased number of spleen cells generating MuLV, compared to controls treated either with purified spleen fraction or with diluent (buffered salt solution). More than two doses of thymic extract (up to nine, given every 2 days after infection) had no further effect on plasma virus titer or number of splenic infectious centers. Treatment with either thymic or splenic extracts increased the incidence of virus-related membrane antigen appearing on the surface of spleen cells, compared to diluent-treated control animals. These results indicate that thymosin may enhance infection by or replication of murine oncornaviruses. These observations support the concept that infection and alteration of lymphoreticular cells with murine oncornavirus occur more readily in cells that have reached a specific level of differentiation and/or activation and that this stage of development can be brought about by agents such as thymic product.

2651 ENHANCEMENT OF INFECTIVITY AND ONCOGENICITY OF A MURINE LEUKEMIA VIRUS IN ADULT MICE BY X-IRRADIATION. (Eng.) Levy, R. L. (Scripps Clinic and Res. Foundation, La Jolla, CA 92037); Lerner, R. L.; Dixon, F. J. *Cancer Res* 36(6): 2090-2095; 1976.

To define the factors involved in the oncogenic effects of transferred murine leukemia virus (MuLV) in adult mice, 12 adult BALB/c mice were injected with the virus, which induced thymic lymphomas histologically indistinguishable from those caused by neonatal infection. X-irradiation permitted early and high levels of viral expression when given before or after MuLV administration and hastened the development of lymphomas. Expression of virus was assayed by a radioimmune assay for murine p30. Seventeen to 21 days after infection, there was 0.3 μg p30/ml serum, approximately 5 times normal. In the x-irradiated (600 R) mice, there were 2.7 μg p30/ml serum. The virus produced by infected adult mice was infectious and oncogenic when given to newborn mice. Cellular reconstitution appeared to lower the expression of virus after irradiation and MuLV infection.

2652 SUPPRESSION OF MURINE TYPE-C RNA VIROGENES BY TYPE-SPECIFIC ONCORNAVIRUS VACCINES: PROSPECTS FOR PREVENTION OF CANCER. (Eng.) Huebner, R. J. (Lab. RNA Tumor Viruses, Natl. Cancer Inst., Bethesda, MD 20014); Gilden, R. V.; Lane, W. T.; Toni, R.; Trimmer, R. W.; Hill, P. R. *Proc Natl Acad Sci USA* 73(2): 620-624; 1976.

Immunization of crossbred AKR, C57L, SWR, NIH Swiss and Balb/cCan mice and their F_1 progeny with combined killed and live Gross leukemia virus AKR type C viral vaccines suppressed endogenous N-type AKR virus up to 10,000-fold during the first 30-118 days of life. Since previous studies in the same and similar crossbred systems revealed direct correlations between low and high levels of type C virus early in life with low and high incidences of leukemia and other cancers later in life, it is believed that prospects for suppression of spontaneous neoplasms are good. However, 8-14 mo will be required to achieve the final results. The prevention of cancers by serotype-specific vaccines would provide conclusive proof of endogenous viral etiology.

2653 PHENOTYPIC VARIANTS OF DIFFERENTIATION-INDUCIBLE FRIEND LEUKEMIA LINES: ISOLATION AND CORRELATION BETWEEN INDUCIBILITY AND VIRUS RELEASE. (Eng.) Ikawa, Y. (Lab. Viral Oncology, Cancer Inst., Tokyo 170, Japan); Inoue, Y.; Kameji, R.; Aida, M.; Shibata, C.; Sugano, H. *Bibl Haematol (Basel)* (43): 37-47; 1976.

Clones of cultured Friend leukemia cells whose differentiation potential varied in response to dimethylsulfoxide (DMSO) are described. Some clones were superinducible, while others were resistant. Some of the clones resistant to in vitro differentiation induction by DMSO did show some evidence of differentiation in the ip diffusion chamber culture, and one clone noninducible by DMSO became inducible by the further addition of Newcastle disease virus interferon. A superinducible clone showed mRNA synthesis preceding Hb synthesis, whereas a differentiation-resistant clone showed only insignificant leakage of globin gene, suggesting a transcriptional control of the globin gene in this line. There was no correlation between the amount of type C virions released from cells and their differentiation inducibility. Potent Friend leukemia virus was harvested from hosts with increased erythroid precursor cells, suggesting host cell dependency of spleen focus-forming activity of the virus. The role of the cell membrane in differentiation inducibility is discussed. (11 references)

2654 ERYTHROID DIFFERENTIATION IN CULTURED FRIEND LEUKEMIA CELLS TREATED WITH METABOLIC INHIBITORS. (Eng.) Ebert, P. S. (Viral Biology Branch, Natl. Cancer Inst., Bethesda, MD 20014); Wars, I.; Buell, D. N. *Cancer Res* 36(5): 1809-1813; 1976.

Cultured Friend leukemia cells were treated with a variety of compounds to determine whether drugs other than dimethylsulfoxide are capable of inducing erythroid differentiation. Several hormones, inhibitors of RNA synthesis, organic solvents, inhibitors of DNA polymerase, sulfhydryl inhibitors, and inducers of δ-aminolevulinic acid synthetase administered singly did not stimulate hemoglobin synthesis. However, inhibitors of DNA and RNA synthesis such as adriamycin, mitomycin C, and hydroxyurea:mithramycin were synergistic in stimulating erythroid differentiation.

2655 NUCLEAR CHROMATIN CHANGES DURING ERYTHROID DIFFERENTIATION OF FRIEND VIRUS INDUCED LEUKEMIC CELLS. (Eng.) Darzynkiewicz, Z. (Sloan-Kettering Memorial Cancer Center, New York, NY 10021); Traganos, F.; Sharpless, T.; Friend, C.; Melamed, M. R. *Exp Cell Res* 99(2): 301-309; 1976.

Acridine orange (AO) binding to DNA and thermal denaturation of DNA in situ were studied in Friend leukemic (FL) cells that had undergone erythroid differentiation by dimethylsulfoxide (DMSO) and in nondifferentiated FL cells using flow-through cytofluorometry. The differentiated cells bound less AO than did the nondifferentiated ones. The difference in AO binding was higher in the spectrum of emission characteristic for AO intercalation (at 530 nm) than for AO stacking (>600 nm) and depended on AO concentration. The difference was abolished after extraction of acid-soluble macromolecules from cells. During stepwise extraction by lowering pH, there was a progressive increase of AO binding to DNA. Most of the increase in AO binding of the nondifferentiated cells occurred at pH 2.5-2.0, and that of the differentiated cells occurred at pH 1.75-1.50. There were differences in sensitivity of DNA in situ to heat denaturation between differentiated and nondifferentiated cells, as evidenced by the variation in height and position of the melting bands on derivative melting profiles. The changes described suggest that a profound modulation of chromatin structure, perhaps involving altered DNA-histone interactions, occurs during the DMSO-induced erythroid differentiation of FL cells. These changes are related to the final stages of differentiation, as fully differentiated cells (those cultured with DMSO for 4-6 days) are already engaged in translation of the globin message.

2656 IMMUNOSUPPRESSION BY MOLONEY LEUKEMIA VIRUS: LACK OF CORRELATION BETWEEN VIRUS REPLICATION AND THE IMMUNOSUPPRESIVE EFFECT. (Eng.) Cerny, J. (Harvard Univ. Sch. Public Health, Boston, MA 02115); Proffitt, M. R.; Essex, M. *J Natl Cancer Inst* 56(4): 819-822; 1976.

Young adult mice were infected with 10^4 plaque-forming units (PFU) of Moloney murine leukemia virus (M-MuLV) either obtained from several serial passage in mice (animal passage virus, AP) or harvested after three in vitro passages of the AP M-MuLV in fibroblasts (tissue culture virus, TC). Replication of TC and AP M-MuLV in spleen cells was determined by an infectious center (IC) assay at 1 and 2 wk after the infection. Immune responsiveness of spleen cells was evaluated in challenge with SRBC and subsequent enumeration of antibody plaque-forming cells (PFC). TC M-MuLV replicated faster in the spleen than did AP M-MuLV and reached about 10- to 100-fold higher titers. However, the response of anti-SRBC PFC, suppressed to the same degree in the spleens of mice infected with TC or AP virus, was about 10% to 50% of the control response. A comparison of virus replication with the anti-SRBC response in aliquots from the same spleens showed no correlation between virus IC and antibody PFC. Both TC and AP M-MuLV induced the expression of virus-specific, cell membrane antigen on spleen cells. These findings indicate that virus replication and the immunosuppressive effect of this virus may be unrelated functions that can be expressed independently.

2657 A VIRALLY INDUCED OSTEOSARCOMA IN RATS: A MODEL FOR IMMUNOLOGICAL STUDIES OF HUMAN OSTEOSARCOMA. (Eng.) Friedlaender, G. E. (Naval Medical Res. Inst., Bethesda, MD 20014); Mitchell, M. S. *J Bone Joint Surg* 58-A(3): 295-302; 1976.

Inoculation of Moloney sarcoma virus into the medullary canal of the tibia in newborn Wistar-Lewis rats resulted in an initially localized osteosarcoma that usually metastasized to the lung and resulted in the death of the animal within 4-5 wk. The tumor cells were grown in tissue culture and used as target cells in the assay of lymphocyte-mediated cytotoxicity using a microcytotoxicity and a radioisotope labeling method. Lymphocyte-mediated cytotoxicity was demonstrated throughout the course of the clinical disease as well as in a same number (15 of the 75 surviving more than 2 wk) of animals that showed spontaneous regression of their tumors. Serum factors that could block or augment the cellular response were also identified. This model resembles the spontaneous osteosarcoma of humans in many respects and may be useful for studies of the human disease.

2658 CHARACTERIZATION OF VIRUS-FREE GUINEA PIG TUMORS INDUCED BY KIRSTEN SARCOMA VIRUS.

(Eng.) Rhim, J. S. (Microbiological Associates, Inc., Bethesda, MD 20014); Cho, H. Y.; Kim, J. M.; Green, I. *J Natl Cancer Inst* 56(6): 1233-1236; 1976.

Tumors were induced by Kirsten sarcoma virus (KiSV) in inbred strain 13 guinea pigs. The tumor cells were established in culture and characterized. The KiSV-induced sarcoma cells were virus-free and nonproducing; however, they contained a rescuable sarcoma genome. A type B guinea pig retravirus was readily activated from the tumor cells after induction with 5-bromodeoxyuridine (BUdR). The particles, approximately 100 nm in the mature form, had a density of 1.16 to 1.17 g/ml and contained reverse transcriptase activity; however, they were not truly typical of type B or type C viruses. BUdR induction of guinea pig retravirus was further enhanced by treatment with dexamethasone, a synthetic glucocorticoid hormone. (28 references)

2659 RETRANSFORMATION OF A THERMOSENSITIVE BALB/C-3T3 TRANSFORMANT BY MURINE SARCOMA VIRUS AT THE NON-PERMISSIVE TEMPERATURE. (Eng.)

Kamely, D. (Natl. Heart and Lung Inst., Natl. Insts. Health, Bethesda, MD 20014). *Nature* 261(5555): 50-52, 1976.

An independent temperature-sensitive clone of BALB/c-3T3 cells (tsCl.1) was infected at the nonpermissive temperature (39 C) with a Kirsten strain of murine sarcoma virus (MSV) to determine whether the apparent loss of the ability of the transformants to express a transformed phenotype at the nonpermissive temperature is reversible. A method involving exposure of virus-infected cultures to ^{3}H-thymidine in a medium containing low serum concentration (0.5%-1% fetal calf serum) was used to screen large numbers of cultures for transformed foci on day 7 of culture. The infected clone demonstrated growth in soft agar, increase in caseinolytic activity, higher saturation densities, and loss of regulation of growth and DNA synthesis. The results therefore suggest that murine sarcoma virus-induced transformation of temperature-sensitive cell variants can be accomplished at the nonpermissive temperature.

2660 CHARACTERIZATION OF RAT GENETIC SEQUENCES OF KIRSTEN SARCOMA VIRUS: DISTINCT CLASS OF ENDOGENOUS RAT TYPE C VIRAL SEQUENCES. (Eng.)

Scolnick, E. M. (Tumor Virus Genetics Branch, Natl. Cancer Inst., Bethesda, MD 20014); Goldberg, R. J.; Williams, D. *J Virol* 18(2): 559-566; 1976.

To determine the relationship between at least one portion of the rat-specific information of the Kirsten sarcoma virus (Ki-sv) or the Harvey sarcoma virus (Ha-sv) and endogenous type C virus genetic information in various rat cells, the Ki-sv genome was characterized by hybridization to virus-specific complementary DNA (cDNA). The homologous sequences were present in multiple copies per diploid rat cellular genome in a variety of different rat cellular DNAs. In certain cells that constitutively express only low levels of sequences homologous to Ki-sv, bromodeoxyuridine treatment led to the expression of high levels of these sequences in RNA. Supernatants from cell lines producing the sequences homologous to Ki-sv contained high levels of these sequences, which are purified to the same degree as the previously known rat type C viral nucleic acid sequences by type C particles being released from such cells. The results indicate that the sequences in rat cells homologous to Ki-sv and Ha-sv have three characteristics of known mammalian type C viruses, and suggest that at least part of the Ki-sv rat-derived sequences represent a distinct class of endogenous rat type C virus that has no detectable homology to the other known class.

2661 VIRUS-INDUCED OSTEOSARCOMA IN RATS. (Eng.)

Czitrom, A. A. (Univ. Toronto, Ontario, Canada); Pritzker, K. P. H.; Langer, F.; Gross, A. E.; Luk, S. C. *J Bone Joint Surg* 58-A(3): 303-308; 1976.

An experimental model for osteosarcoma was developed in which, after Moloney murine sarcoma virus was injected into the tibial marrow space of Fischer, Wistar-Lewis, and ACI inbred neonatal rats, a highly malignant neoplasm arose within 10 days. This tumor was readily maintained in tissue culture and was transplantable to adult rats. It developed in the metaphyseal marrow of several bones, was locally invasive, metastasized to the lungs, and histologically resembled osteosarcoma.

2662 ESTABLISHMENT AND CHARACTERIZATION OF CELL LINES FROM GROSS VIRUS-INDUCED RAT LEUKEMIA. (Eng.)

Nishiyama, T. (Faculty Medicine, Kyushu Univ., Fukuoka 812, Japan); Okano, H. *Z Krebsforsch* 86(1): 13-22; 1976.

Light and electron microscopic investigation of the individual or mixed cultivation of cells from thymus and/or bone marrow tissues of either normal or Gross virus-infected W/Fu rats revealed four continuous cell lines: (a) individual cultivation of the cells from thymic lymphoma, (b) combined cultivation of cells from thymic lymphoma and normal bone marrow, (c) bone marrow of a leukemic lymphoma and normal thymus, and (d) bone marrow of a leukemic rat supplemented with cell-free extracts of normal thymus homogenates. All four cell lines had similar morphological features. They corresponded morphologically to lymphoblasts, grew rapidly in suspension, and were transplantable in newborn rats. The cells released abundant type C virus particles. The inoculation of cell-free culture fluid into newborn rats resulted, after 7-9 wk, in a high incidence of thymic lymphoma.

2663 SPONTANEOUS RELEASE OF ENDOGENOUS ECOTROPIC TYPE C VIRUS FROM RAT EMBRYO CULTURES.

(Eng.) Rasheed, S. (Univ. Southern California Sch. Medicine, Los Angeles, CA 90033); Bruszewski, J.; Rongey, R. W.; Roy-Burman, P.; Charman, H. P.; Gardner, M. B. *J Virol* 18(2): 799-803; 1976.

Type C viruses were isolated from embryo cultures of Sprague-Dawley and Fischer rats. Both viruses (termed rat leukemia virus, RaLV) were released spontaneously from rat embryo cells, had a density of 1.14 to 1.15 g/cm^3 based on equilibrium sedimentation in sucrose gradients, contained 60S-70S RNA, RNA-directed DNA polymerase, and rat type C virus-specific 30,000 molecular wt protein determinants. Molecular hybridization studies with the Sprague-Dawley RaLV 60S-70S RNA showed that the virus-specific nucleotide sequences were present in the DNA of rat embryos. Both Sprague-Dawley and Fischer RaLV rescued the murine sarcoma virus genome from Kirsten murine sarcoma virus-transformed nonproducer cells and were neutralized by antisera to the RPL strain of RaLV. In contrast to previous RaLVs, these viruses propagated in their own cells of origin as well as in cells of heterologous rat strains. This spontaneous release of endogenous type C viruses may have been influenced by an environmental or genetic change in the breeding stock or by a particularly early embryonic age.

2664 INFLUENCE OF GLUCOCORTICOID, ESTROGEN, AND ANDROGEN HORMONES ON TRANSFORMATION OF HUMAN CELLS IN VITRO BY FELINE SARCOMA VIRUS.

(Eng.) Schaller, J. P. (Coll. Veterinary Medicine, Ohio State Univ., Columbus, OH 43210); Milo, G. E.; Blakeslee, J. R., Jr.; Olsen, R. G.; Yohn, D. S. *Cancer Res* 36(6): 1980-1987; 1976.

The effects of various corticosteroid, androgen, and estrogen hormones on the qualitative and quantitative transformation of human cells by feline sarcoma virus (FeSV) were evaluated. Human foreskin cells (D-550) were infected in vitro by the Snyder-Theilen strain of feline sarcoma virus (FeSV). This infection produced small but countable foci and demonstrated ''single-hit'' dose-response kinetics. Significant quantitative and qualitative enhancement of focus formation was observed when the glucocorticoid hormones dexamethasone, hydrocortisone, cortisol acetate, and prednisolone were added to cell cultures (1.0 μg/ml) 24 hr postinfection. However, aldosterone, while inducing qualitatively larger foci, did not bring about a quantitative enhancement in total foci number. By contrast, the nonglucocorticoid hormones 17β-estradiol, progesterone, cortisone acetate, methyltestosterone, and estrone elicited little or no effect on focus induction by FeSV. These results suggest that a posttranscriptional effect modulates viral genome expression, resulting in an increased efficiency of viral transformation and an increased proliferation of transformed cells.

2665 PURIFICATION AND PROPERTIES OF A VIRION PROTEIN KINASE.

(Eng.) Silberstein, H. (Albert Einstein Coll. Medicine, Bronx, New York, NY 10461); August, J. T. *J Biol Chem* 251(10): 3176-3184; 1976.

The protein kinase associated with virions of frog virus 3, assumed to be a functional component of the infective process, was purified to apparent homogeneity by ion exchange chromatography and gel filtration. The enzyme protein appeared as a single polypeptide of molecular wt 50,000 to 55,000 as determined by gel filtration, glycerol gradient sedimentation, and sodium dodecyl sulfate-polyacrylamide gel electrophoresis. It comprised approximately 0.4% of the total virion protein. The activity was classified as a cyclic nucleotide-independent protein kinase, as it was not affected by cyclic adenosine 3':5'-monophosphate or cyclic guanosine 3':5'-monophosphate, nor inhibited by a cyclic nucleotide-dependent protein kinase inhibitor protein; it utilized GTP as well as ATP as a phosphate donor. The greatest rates of phosphorylation were obtained with acidic phosphoprotein substrates such as casein or phosvitin, although potential physiological substrates for this activity included specific virion polypeptides of frog virus.

2666 CHARACTERIZATION OF A VIRION PROTEIN KINASE AS A VIRUS-SPECIFIED ENZYME.

(Eng.) Silberstein, H. (Albert Einstein Coll. Medicine, Bronx, New York, NY 10461); August, J. T. *J Biol Chem* 251 (10): 3185-3190; 1976.

In order to study frog virus protein kinase, antibodies that completely inhibited its enzymatic activity were obtained by immunization of rabbits with the purified enzyme. This inhibition provided a specific probe for the frog virus protein kinase, since the antiserum had no inhibitory effect on a variety of other protein kinases, including the activity of uninfected cells, or the protein kinase associated with vesicular stomatitis virus or vaccinia virus cultivated in the same cell line as frog virus. The frog virus protein kinase was characterized as a virus-specified protein on the basis of the following observations: (a) the virion protein kinase was antigenically distinct from essentially all of the protein kinase expressed in uninfected cells; (b) following infection by frog virus more than a 15-fold increase was detected in the specific activity of intracellular protein kinase, mostly related to the virion enzyme; (c) when frog virus was grown in cells derived from widely different species, the antigenic and biochemical specificities of the virion protein kinase remained identical; and (d) screening of cells infected with different temperature-sensitive mutants of frog virus indicated that certain viral mutants failed to synthesize this protein kinase when cultivated at the nonpermissive temperature. This report supports the hypothesis that in the case of frog virus, the protein kinase is virus specified.

2667 IMMUNOLOGICAL ANALYSIS OF STRUCTURAL POLYPEPTIDES OF WOOLLY MONKEY-GIBBON APE TYPE C VIRUSES. EXPRESSION OF WOOLLY MONKEY HELPER VIRAL ANTIGENS IN CELLS TRANSFORMED BY WOOLLY MONKEY SARCOMA VIRUS.

(Eng.) Aaronson, S. A. (Natl. Cancer Inst., Bldg. 37, Bethesda, MD 20014); Stephenson, J. R.; Tronick, S. R.; Hino, S. *Bibl Haematol (Basel)* (43): 102-109; 1976.

Immunological properties of three structural polypeptides of a group of type C viruses isolated from the woolly monkey (WMV) and gibbon ape (GAV) are described. WMV and GAV competed with similar efficiencies in competition immunoassays developed for the p30 of the WMV using either antiserum against homologous WMV or one of the GAVs. In a competition immunoassay utilizing antiserum to WMV to bind ^{125} WMV p12, only WMV competed efficiently. In reciprocal studies using an immunoassay for the p12 of one of the GAVs, only the homologous virus completely inhibited binding of ^{125}I-GAV p12 by antiserum to the GAV; neither the WMV nor a second GAV isolate demonstrated more than partial cross-reactivity in this assay. In a heterologous immunoassay utilizing limiting antiserum prepared against the GAV to precipitate ^{125}I-WMV gp70, the WMV and two GAV isolates reacted efficiently, but when the ^{125}I-WMV gp70 was used in combination with antibody to the WMV, only the homologous virus was able to compete efficiently. When an antiserum against FeLV was used to precipitate WMV gp70, a more broadly reactive class of antigenic determinants was measured, with cross-reactivity not only to WMV and GAV, but also to Rauscher murine leukemia virus, AKR murine leukemia virus, and feline leukemia virus. Antigenic determinants detected by direct precipitation of viral gp70 were more broadly reactive than those determinants responsible for virus neutralization. Immunoassays for WMV p12, p30, and gp70 detected high levels of all three polypeptides in NRK cells chronically infected with woolly monkey helper virus, but uninfected NRK cells were not reactive in these assays. Three distinct patterns of immunological reactivity were observed in cells transformed by woolly sarcoma virus (WSV): two clonal lines demonstrated no greater reactivity than NRK cells; one line demonstrated high levels of viral p12 in the absence of either WMV p30 or gp70 expression; and the majority of transformants demonstrated high levels of both WMV p12 and p30 but lacked gp70. To determine whether these differences were virus- or cell-specific, WSV was rescued from each transformed phenotype and introduced into new NRK cells. Similar reactivities developed, indicating viral specificity. (36 references)

2668 A COMPARATIVE EVALUATION OF METHODS FOR ISOLATION OF RNA-DIRECTED DNA POLYMERASE FROM CELLS IN A RECONSTITUTED SYSTEM.

(Eng.) Allaudeen, H. S. (Sch. Medicine, Yale Univ., New Haven, CT 06511); Sarngadharan, M. G.; Gallo, R. C. *Biochim Biophys Acta* 435(1): 45-62; 1976.

The relative merits of several procedures for the isolation of RNA-directed DNA polymerase from cells by the use of a reconstituted model system consisting of a mixture of wooly monkey (simian) sarcoma virus and a cultured human lymphoblastoid cell line, NC-37, were compared. When the cell-virus mixture was gently disrupted and fractionated by differential centrifugation, most of the added polymerase was recovered associated with a particulate fraction obtained from the postmitochondrial supernatant. Purification of the polymerase was best achieved starting from this fraction. The particulate fraction itself could be purified by gel filtration through a Sepharose 2 B column. This procedure did not significantly alter the composition of viral and cellular DNA polymerases. Although as little as 7.5 x 10^5 viral particles were sufficient to detect RNA-directed DNA polymerase activity, a minimum of about 10^{11} particles was necessary for the isolation and unequivocal characterization of the enzyme from the cell-virus mixture by subcellular fractionation and chromatographic separation from cellular DNA polymerases. Purified RNA-directed DNA polymerase had the same primer-template characteristics, sedimentation properties, and immunological cross-reactivity as the enzyme purified from density gradient-banded virions of simian sarcoma virus. Methods involving total extraction of the cell-virus mixture either by repeated freezing and thawing followed by detergent treatment or by Dounce homogenization and treatment with high salt and detergent failed to provide RNA-directed DNA polymerase free of cellular DNA polymerases. Because of this, low levels of the enzyme may be missed when these approaches are used.

2669 RD-114, BABOON, AND WOOLLY MONKEY VIRAL RNAs COMPARED IN SIZE AND STRUCTURE.

(Eng.) Kung, H. J. (Dept. Chemistry, California Inst. Technology, Pasadena, CA 91125); Hu, S.; Bender, W.; Bailey, J. M.; Davidson, N.; Nicolson, M. O.; McAllister, R. M. *Cell* 7(4): 609-620; 1976.

The molecular wt, subunit composition, and secondary structure patterns of the RNAs from an endogenous baboon virus and from a woolly monkey sarcoma virus were examined and compared to the properties of the RNA of RD-114, an endogenous feline virus. The high molecular wt RNA extracted from each of these three viruses had a sedimentation coefficient of 52S and a molecular length, measured by electron microscopy, of 16-20 kb (kilobase; 1000 nucleotides). Each such RNA was a dimer, containing two monomer subunits 8-19 kb in length (molecular wt 3 x 10^6 daltons). The two monomer subunits were joined at their nonpoly(A) ends in a structure called the dimer linkage structure. The dimer linkage dissociated at about 87 C in aqueous 0.1 M Na+ for RD-114 and baboon viral RNAs, but at 66 C for woolly monkey RNA. All three viral RNAs had two large loops of similar size and position symmetrically placed on either side of the dimer linkage structure. Since the baboon virus is partially related to RD-114, and the woolly monkey virus is unrelated to either of the other two, the dimer linkage and symmetrical loops are surprisingly similar and may well be common features of type C virus RNAs.

2670 PROTEINS OF YABA MONKEY TUMOR VIRUS I. STRUCTURAL PROTEINS.

(Eng.) Fenger, T. (Dept. Microbiology, Southern Illinois Univ., Carbondale, Il 62901); Rouhandeh, H. *J Virol* 18(2): 757-764; 1976.

To define the number of polypeptide species of the purified Yaba virion and to locate these polypeptides within the specific structures of the virion, they were studied by polyacrylamide gel electrophoresis. Electrophoresis of Yaba virions (proteins) dissociated by sodium dodecyl sulfate and 2-mercaptoethanol in continuous and discontinuous buffer systems yielded

37 polypeptide species by staining and by counting bands of radioactively labeled polypeptides. The molecular wt of the viral polypeptide species ranged from 10,000 to 220,000 when the relative distance of migration of viral proteins was compared with proteins of known molecular wt. Two polypeptides were removed from purified virions by nonionic detergent nonidet P-40 treatment, and the amount of one polypeptide was reduced. Twenty-one polypeptide species, none of which was labeled with radioactive glucosamine, were constituents of the core. The other 16 formed the lateral bodies and envelope.

2671 NUCLEIC ACID AND PROTEIN STUDIES OF THE MASON-PFIZER VIRUS. (Eng.) Schlom, J. (Natl. Cancer Inst., Bldg. 37, Room 1B19, Bethesda, MD 20014); Colcher, D.; Drohan, W.; Schochetman, G. *Bibl Haematol (Basel)* (43): 134-140; 1976.

The characterization of the protein, glycoprotein, and RNA of Mason-Pfizer virus (MPV) is described. The major viral polypeptide has a molecular wt of 27,000 (p27). Five other proteins are also detected: p10, p12, p14, p20, and p68. The major glycoprotein is gp68; occasionally some gp75 was observed that may or may not represent the same glycoprotein. A small amount of gp20 is also present. The protein makeup is similar in viruses from various cells, indicating that they are coded for by the virion. Two glycoproteins, gp36 and gp48, represent major portions of the glycoproteins of MPV grown in NC-37 cells, but they are not present in MPV grown in rhesus foreskin cells. The major external protein of the virion is gp68. The RNA structure in MPV is similar to that of murine leukemia virus in terms of its molecular wt (8×10^6) and that of its subunits (2.8×10^6). There are no detectable differences in nucleic acid sequences among the RNA's of MVP and x381 (a virus from rhesus-lactating mammary gland cultures) and RNA isolated from the cytoplasm of HeLa and AO OH cell lines producing particles morphologically similar to MPV. The lack of appreciable hybridization of MPV ^{125}I-RNA to the DNA of normal NC-37 cells and two clones of HeLa cells indicates that MPV is not an endogenous virus of human cells; i.e., the entire genome is not present as a provirus in the DNA of all cells of the species. MPV was also not found to be endogenous in 11 nonprimate mammalian species. (19 references)

2672 DISTRIBUTION OF INGESTED FERRITIN AND ACID PHOSPHATASE ACTIVITY IN THE VACUOLAR SYSTEM OF NORMAL AND VIRUS-TRANSFORMED MOUSE 3T3 CELLS. (Eng.) Beadle, D. J. (Sch. Biological Sciences, Thames Polytechnic, Wellington St., London SE18 6PF, Great Britain); Dawson, A. L.; Amos, S.; Black, P. *Cytobios* 13(50/55): 141-149; 1975.

The vacuolar system of normal and transformed mouse fibroblasts was investigated with ferritin as an exogenous marker and acid phosphatase as an endogenous marker. Ferritin particles were found in dense and residual bodies; both of these structures exhibited acid phosphatase activity. Ferritin was also found in vesiculotubular elements. Acid phosphatase was associated with the Golgi apparatus and primary lysosomes. Non-proliferative confluent cultures of normal cells retained ferritin within the vacuolar system for at least 8 days, whereas proliferative cultures of both normal and transformed cells cleared it within 48 to 72 hr. Normal mouse fibroblasts apparently endocytose more rapidly than the transformed counterparts.

2673 MAPPING OF POLY(A) SEQUENCES IN THE ELECTRON MICROSCOPE REVEALS UNUSUAL STRUCTURE OF TYPE C ONCORNAVIRUS RNA MOLECULES. (Eng.) Bender, W. (Div. Biology, California Inst. Technology, Pasadena, CA 91125); Davidson, N. *Cell* 7(4): 595-607; 1976.

A convenient electron microscope label for mapping poly(A) sequences was synthesized. Short lengths of poly(dT) were polymerized onto nicked circular SV40 DNA with the enzyme terminal deoxynucleotidyl transferase. An RNA or DNA molecule of interest was treated with glyoxal, hybridized briefly with the poly(dT) circles, and spread for microscopy; poly(A) stretches were clearly marked because they were attached to the poly(dT) on the easily recognized SV40 duplex circles. The RNAs of several type C oncornaviruses were examined by this method. The endogenous feline virus (RD-114), the endogenous baboon virus (BKD), and the woolly monkey sarcoma virus (WoMV) all contained a dimer of RNA subunits held together in a central secondary structure feature (dimer linkage structure). Both ends distal to the dimer linkage structure hybridized to the SV40-poly(dT). If it is assumed that both poly(A)s were on the 3' ends of the subunits and that both subunits are identical, the two identical subunits were held together by interactions between sequences close to the 5' ends.

2674 PREFERENTIAL SYNTHESIS OF VIRAL LATE RNA BY NUCLEI ISOLATED FROM SV40 LYTICALLY INFECTED CELLS. (Eng.) Gilboa, E. (Dept. Biochemistry, Weizmann Inst. Science, Rehovot, Israel); Aviv, H. *Cell* 7(4): 567-573; 1976.

Nuclei from SV40-infected monkey cells were isolated late in lytic infection, and their cell-free transcriptional activity was characterized. ^{3}H-RNA synthesized in vitro was hybridized to excess quantities of separated SV40 DNA strands that were each covalently bound to Sepharose. Of the newly synthesized RNA, 3-5% was virus-specific, and plus-strand DNA, coding for late RNA sequences, was transcribed at a rate about 15 times higher than that of the minus-strand DNA, which codes for early RNA sequences. This indicates that transcriptional control has a major role in determining the relative abundancy of early and late RNA classes in lytically infected cells.

2675 CELL-SURFACE ANTIGENS OF DNA VIRUS (SV40) INFECTED AND TRANSFORMED CELLS. (Eng.) Tevethia, S. S.; Tevethia, M. J. In: *Cellular Membranes and Tumor Cell Behavior. A Collection of Papers Presented at the Twenty-Eighth Annual Symposium on Fundamental Cancer Research, 1975.* University of

Texas System Cancer Center, M. D. Anderson Hospital and Tumor Institute. (Baltimore: Williams and Wilkins Co.): pp. 447-467; 1975.

The properties of SV40-specific transplantation antigen (TSTA) and of other cell-surface antigens and the role of TSTA in tumorigenesis by the oncogenic DNA virus SV40 are reviewed. The presence of TSTA in SV40-transformed cells has been demonstrated by transplantation rejection tests. In addition to the macrophage migration inhibition test, the cellular immune response to TSTA can also be measured with the in vivo tumor cell neutralization test. Serological tests (direct immunofluorescence, mixed hemadsorption, colony inhibition, isotopic antiglobulin, and cytotoxic tests) have demonstrated the presence of specific and fetal antigens at the surface of SV40-transformed cells. While TSTA is specific for SV40, embryonic antigens cross-react from tumor to tumor and with fetal tissue. The target cell involved in TSTA induction was investigated by culturing normal lymphocytes on top of a monolayer of cells infected by SV40. The lymphocytes sensitized on SV40-infected macrophages and on Balb/3T3 cells, were able to inhibit SV40 tumor cells in vivo, indicating that these mouse cells synthesized TSTA. TSTA mediates the development of the cellular immune response in the host, which leads to the rejection of developing tumors. In this process, normal macrophages cooperate with T-lymphocytes. The most likely explanation of the growth of SV40 tumors in the nonpermissive host in the presence of a cellular immune response to TSTA is that the TSTA is released by the transformed cells in a soluble form and is able to block the activity of sensitized T-lymphocytes, thereby allowing the tumor to grow. The apparent nononcogenicity of the virus in its natural host may result from the fact that SV40 induces TSTA in infected cells to which the host becomes sensitized. The infected cells eventually release progeny and die. Subsequently, each time the progeny viruses infect cells, synthesis of TSTA occurs, and the cellular immune response of the host is initiated and potentiated. This initiation and potentiation of the host response to TSTA may be responsible for eliminating potentially transformed cells.

2676 THE SOLUBILITY CHARACTERISTICS OF SV40 TUMOR ANTIGEN. (Eng.) Kellermayer, M. (Baylor Coll. Medicine, Houston, TX 77025); Talas, M.; Wong, C.; Busch, H.; Butel, J. S. *Exp Cell Res* 99(2): 456-460; 1976.

The solubility characteristics of SV40 tumor (T) antigen were investigated. Cell nuclei were isolated from cover glass cultures of SV40-infected or transformed cells by detergent treatment. Treatment in a buffered isotonic sucrose solution and immunofluorescence indicated that the isolated nuclei contained typical SV40 T antigen. Dissolving the detergent in an isotonic electrolyte solution containing 0.15 M monovalent cations (K^+) resulted in the extraction of the T antigen from the nuclei. Complement fixation tests demonstrated that the various solutions had no deleterious effects on the antigenicity of T antigen. Under identical conditions, the SV40 capsid (V) antigen was not extracted from infected cell nuclei. These solubility characteristics placed SV40 T antigen in the class of ''loosely bound'' nuclear proteins, which includes nonhistone proteins that appear to have cell regulatory functions. These results suggest that the biological function of T antigen may be regulatory in nature.

2677 REVERSE TRANSCRIPTASE ACTIVITY ASSOCIATED WITH R-TYPE VIRUS-LIKE PARTICLES OF SV40-TRANSFORMED HAMSTER CELLS (TSV$_5$ CLONE 2). (Eng.) Rossignol, J.-M. (Institut de Recherches Scientifiques sur le Cancer, Villejuif, France); Kress, M.; De Recondo, A.-M. *Intervirology* 5(5): 273-292; 1975.

R-type virus-like particles (VLP) were partially isolated from SV40-transformed hamster cells (TSV$_5$ clone 2) and assayed for reverse transcriptase activity. Confluent cells were disrupted by cavitation. The disrupted cells were centrifuged at 20,000 g for 15 min, and the resulting supernatant centrifuged for 45 min at 105,000 g. R-type VLP were found in the cytoplasm, and were frequently localized within ergastoplasmic cisternae, but never in the nucleus. R-type VLP were also found in the 105,000 x g pellet, which contained at least two DNA polymerase activities. One was DNA-dependent, and easily removed from the pellet by a second wash with buffer followed by a second centrifugation for 45 min at 105,000 x g. The other DNA polymerase activity was hybrid-dependent and was only released from the pellet by a treatment with the detergent NP40 (0.1%). The 105,000 x g supernatant was pooled with NP40-solubilized proteins from this pellet and chromatographed on phosphocellulose. Three peaks were eluted, one of which was designated DNA polymerase B and was only hybrid-dependent. The elution molarity of DNA polymerase B was similar to that of the reverse transcriptase of murine oncornaviruses such as Rauscher murine leukemia virus (R-MuLV). In addition, DNA polymerase B could not be distinguished on the basis of its template specificities from the reverse transcriptase of R-MuLV. Efforts to detect a similar DNA polymerase activity in other hamster cells not transformed by SV40 were not successful. DNA polymerase B cannot be distinguished from the reverse transcriptase of R-MuLV by template specificity. The appearance of reverse transcriptase activity associated with R-VLP could be due to the activation of endogenous viruses by SV40 or to a direct action of SV40 on R-VLP.

2678 GENE MAPPING OF SV40: THE BIOLOGICAL ACTIVITY OF SPECIFIC VIRAL DNA FRAGMENTS PRODUCED BY CLEAVAGE WITH HAEMOPHILUS PARAINFLUENZAE RESTRICTION ENDONUCLEASE. (Eng.) Graessmann, A. (Institut fuer Molekularbiologie und Biochemie der Freien Universitaet Berlin, Dl Berlin 33, Arnimallee 22, E. Germany); Graessmann, M.; Bobrik, R.; Hoffmann, E.; Lauppe, F.; Mueller, C. *FEBS Lett* 61(1): 81-84; 1976.

In the attempt to define the region of the simian virus 40 (SV40) genome required for T-antigen synthesis, DNA fragments were prepared by digesting SV40 DNA component I (DNA I) with Haemophilus parainfluenzae I (Hpa I) and parainfluenzae II (Hpa II) restriction endonucleases. The isolated DNA fragments were transferred into permissive monkey cells (TC7) by a macroinjection technique, and T-antigen and viral capsid antigen (V-antigen) synthesis were assayed in these cells. T-antigen synthesis was observed only after microinjection of the SV40 DNA fragment B, which corresponds to 36% of the SV40 genome. Injection of SV40 fragments A, C, and/or D did not induce T-antigen synthesis in TC7 cells. T-antigen formation was first detectable about 10 hr after the microinjection of fragment B, a maximum number of 60% positive cells was seen at 20-24 hr, and no T-antigen was detectable at 80 hr. V-antigen synthesis was not induced by the microinjection of SV40 DNA fragments generated by Hpa I-Hpa II endonuclease cleavage. The results demonstrate that a 40,000-50,000 molecular wt polypeptide coded for by the early part of fragment B contains the determinant group of the T-antigen, and that the C-terminal part of the entire early protein is not required for the T-antigen-antibody reaction.

2679 PRODUCTION OF PLASMINOGEN ACTIVATOR AND COLONIAL GROWTH IN SEMISOLID MEDIUM ARE IN VITRO CORRELATES OF TUMORIGENICITY IN THE IMMUNE-DEFICIENT NUDE MOUSE. (Eng.) Pollack, R.; Risser, R.; Conlon, S.; Freedman, V.; Shin, S.; Rifkin, D. B. In: *Proteases and Biological Control.* Reich, E.; Rifkin, D. B.; Shaw, E., eds. (Cold Spring Harbor, NY: Cold Spring Harbor Lab.): Cold Spring Harbor Conferences on Cell Proliferation, vol. 2, pp. 885-899; 1975.

Factors related to the in vivo loss of growth control, measured as tumorigenicity, in the athymic nude (nu/nu) mouse were investigated. Cells from nonselectively isolated mouse and rat clones arising after infection by simian virus 40 (SV40) were injected into nu/nu mice. The results indicated that the ability to grow as a tumor in these animals is best correlated with loss of the requirement for anchorage. With the use of clones derived by SV40 infection of primary rat embryo cells, it was found that growth without anchorage was correlated with the secretion of plasminogen activator. Sister cultures of primary rat embryo (RE) cells and three SV40-transformed (SVRE) clones were plated in regular medium on plastic dishes and in methylcellulose in four different serum mixtures. In dog and monkey sera, all cultures seemed less adherent to the dish than in fetal calf serum. Growth in semisolid medium was strikingly dependent on the species of serum as well as on plasminogen activator production: primary RE cells did not grow in any of the sera tested, whereas all three SVRE clones grew better in dog or monkey serum than in fetal calf serum. The colonies in methylcellulose were generally solid spheres ranging in size up to 0.3 mm. These results offer indirect evidence that plasminogen activation plays a role in anchorage-dependent cell growth, and they suggest that growth in methylcellulose gel provides an in vitro assay for tumorigenicity. (37 references)

2680 PROPORTIONS AND RENEWAL OF DIFFERENT PHOSPHOLIPIDS IN NORMAL AND SV 40-TRANSFORMED FIBROBLASTS. (Fre.) Maziere, C. (Service de Chimie Biologique, C.H.U. Saint-Antoine, 27, rue Chaligny, 75571 Paris Cedex 12, France); Rampini, C. *Biochimie* 57(10): 1189-1196; 1975.

The phospholipid composition and turnover in hamster fibroblasts (HESV) transformed by the simian virus SV40 were studied at various cell densities. Hamster embryo fibroblasts (HE) and hamster fibroblasts from a cell line that has retained contact inhibition (HEB) served as controls. Cells were seeded at a density of $30,000/cm^2$ in MEM medium with 10% calf serum. The medium was renewed daily. Glucose and antibiotics were added, as was 2.5 μCi/ml ^{32}P phosphate, specific activity 20 mCi/mg. Phospholipid ^{32}P relative to a microgram of cell protein was measured on days 2 and 5 to compare uptake of ^{32}P during exponential and stationary growth. HESV cells had a lower uptake of ^{32}P by phospholipids during exponential growth than did HE and HEB cells, but stationary growth ^{32}P uptake was similar for normal and transformed cells. The ratio of ^{32}P labeled phospholipid to protein decreased for all cell types during stationary growth; however, the decrease for HESV cells was less (25% compared to 50%). The turnover of phosphatides of choline, ethanolamine, and inositol and of diphosphoglycerate and sphingomyelin was measured at intervals of 30 min, 2 hr, and 6 hr on the second day of exponential growth. HESV cells had less sphingomyelin initially and the turnover was slower; diphosphoglycerate was present in higher concentrations in HESV cells and the turnover was greater. Phosphatidylcholine was renewed more frequently than phosphatidylethanolamine in HE and HEB cells, whereas the opposite was true for HESV cells. On the fifth day, when growth was stationary, phospholipid turnover rates decreased and were similar for all three cell types. Normal and transformed cell differences in concentration and turnover of phospholipids may be related to mitochondrial activity or inhibition of cell contact. (28 references)

2681 CONSTRUCTION AND ANALYSIS OF VIABLE DELETION MUTANTS OF SIMIAN VIRUS 40. (Eng.) Shenk, T. E. (Stanford Univ. Sch. Medicine, Stanford, CA 94305; Carbon, J.; Berg, P. *J Virol* 18(2):664-671; 1976.

Mutants that do not affect the viability of simian virus 40 (SV40), with deletions ranging in size from 15 to 200 base pairs, were obtained by infecting CV-1P cells with circularly permuted linear SV40 DNA. The linear DNA was produced by cleavage of closed circular DNA with DNase I in the presence of Mn^2, followed, in some cases, by mild digestion with λ5'- exonuclease. The SV40 map location and the size of each deletion were determined by use of the S1 nuclease mapping procedure and the change in the size of fragments produced by *Hind* II III endonuclease cleavage. Deletions in at least three regions of the SV40 chromosome had slight effect on the rate or yield of viral multiplication and on viral-induced cellular transformation. These regions were located at the following coordinates on the SV40 physical map: 0.17 to 0.18; 0.54 to 0.59; and 0.68 to 0.74. The deletions nearest 0.74 multiplied more poorly than the deletions near 0.68, which were nearly normal. The mutants between 0.54 and 0.59 grew somewhat more slowly and produced slightly lower yields than their wild-type parents; no difference in the amount or distribution of T antigen immunofluorescence was detected. Deletions at the 0.17 to 0.18 area removed sequences at the 3' end of the transcript of both the early and late regions. Studies with additional mutants are needed so that the extent of these dispensable regions can be defined.

2682 STUDIES OF HAMSTER CELLS TRANSFORMED BY ADENOVIRUS 2 AND THE NONDEFECTIVE AD2-SV40 HYBRIDS. (Eng.) Lewis, A. M., Jr.; Breeden, J. H.; Wewerka, Y. L.; Schnipper, L. E. Levine; A. S. In: *Tumor Viruses. Cold Spring Harbor Symposia on Quantitative Biology*. Cold Spring Harbor Lab. (Cold Spring Harbor, NY): vol. 39, pp. 651-656; 1975.

Experiments with adenovirus (Ad)2 and hamster cells indicated that Ad2 did not produce tumors when injected into newborn hamsters. This failure was not corrected by acquiring the portion of the SV40 genome present in any of the nondefective Ad2-SV40 hybrid viruses. However, nonhybrid Ad2 did transform hamster cells in vitro, and these transformed cells produced tumors when injected into newborn hamsters. This finding, and findings from other studies, suggest that the categorization of Ad2 and Ad5 as ''nononcogenic'' viruses is a reflection of host response rather than an intrinsic property of the viruses. Transformation of hamster cells by Ad2 was inefficient, requiring 10^7-10^8 PFU to produce a focus of transformed cells. Transformation of hamster kidney cells by the nondefective hybrids could not be associated with portions of the SV40 genome. During transformation, complex interactions must have occurred between the hamster cell genome and the viral genome of Ad2 or the nondefective hybrids. Thus although there appeared to be very little AD2 DNA in one nonhybrid Ad2-transformed cell line, there was a larger amount of Ad2 DNA in all of the hybrid virus-transformed cell lines. Moreover, although one Ad2+ND_2-transformed line had apparently lost the SV40 portion of the viral genome, it was present and transcribed in another line transformed by the same virus. Finally, in the lines of nondefective hybrid-transformed cells examined thus far, there was extensive transcription from the Ad2 H strand. This pattern of transcription differed from transcription of the viral genome in most nonhybrid AD2-transformed hamster and rat cells in which L-strand transcription predominates.

2683 A NEW SPECIES OF VIRUS-CODED LOW MOLECULAR WEIGHT RNA FROM CELLS INFECTED WITH ADENOVIRUS TYPE 2. (Eng.) Soderlund, H. (Wallenberg Lab., Uppsala Univ., Uppsala, Sweden); Pettersson, U.; Vennstrom, B.; Philipson, L.; Mathews, M. B. *Cell* 7(4): 585-593; 1976.

A virus-coded low molecular wt RNA (5.2S), which migrates slightly faster on polyacrylamide gels than the well characterized adenovirus-specific 5.5S RNA, was isolated from cells infected with adenovirus type 2. Hybridization-competition experiments and RNA fingerprints indicated that the two virus-associated RNAs differ in their primary structures. The gene for 5.2S RNA was located to the right of the gene for 5.5S RNA, on the I strand of a DNA segment extending between positions 30.3 and 32.2 on the map of adenovirus type 2 DNA. Both 5.5S and 5.2S RNA could be detected early after infection and also in the presence of cytosine arabinoside or cycloheximide. After the onset of viral DNA replication, the synthesis of 5.2S RNA leveled off, whereas 5.5S RNA is synthesized in increasing amounts. Both 5.2S and 5.5S RNAs were synthesized in isolated nuclei by an enzyme resembling RNA polymerase III in its sensitivity to α-amanitin. In isolated nuclei, both RNA species were labeled with β ^{32}P-labeled GTP, suggesting that they are initiated at separate promoter sites.

2684 LOW MOLECULAR WEIGHT VIRAL RNAs TRANSCRIBED BY RNA POLYMERASE DURING ADENOVIRUS 2 INFECTION. (Eng.) Weinmann, R. (Div. Biology and Biomedical Sciences, Washington Univ. Saint Louis, MI 63110); Brendler, T. G.; Raskas, H. J.; Roeder, R. G. *Cell* 7(4): 557-566; 1976.

Nuclei isolated from human cells, productively infected with adenovirus 2, synthesized four low molecular wt RNA species that hybridized efficiently to viral DNA. One species corresponds to the 5.5S or virus-associated (VA) RNA, and is designated V_{156}. The other three species are novel and have been designated V_{200}, V_{140}, and V_{130}, since they are approximately 200, 140, and 130 nucleotides in length, respectively. These viral RNAs retain their distinct electrophoretic properties after denaturation with formamide. RNA species with electrophoretic mobilities similar to those of V_{200}, V_{156}, and V_{140} RNAs occurred in the cytoplasmic fraction of cells at late times after adenovirus infection. In isolated nuclei, the four RNAs were all synthesized by DNA-dependent RNA polymerase III, since synthesis is sensitive to high but not to low concentrations of α-amanitin. The synthesis of these low molecular wt RNAs continued for a prolonged period of time in isolated nuclei, suggesting that reinitiation occurs. Adenovirus 2 DNA fragments obtained by digestion with restriction endonucleases *Eco* RI and *Sma* I were used to map the location of the DNA sequences that encode the RNAs. All the low molecular wt RNAs hybridized to a region of the genome between 0.18 and 0.38 fractional length from the left end of the adenovirus genome, suggesting that the respective DNA sequences are clustered. Other nonviral low molecular wt RNAs are synthesized in nuclei isolated from infected cells. These include the cellular 5S rRNA species that was monitored by its hybridization to purified 5S DNA from *Xenopus laevis*.

2685 ANTIGENIC DETERMINANTS OF ADENOVIRUS CAPSIDS. I. MEASUREMENT OF ANTIBODY CROSS-REACTIVITY. (Eng.) Willcox, N. (Natl. Inst. Medical Res., Mill Hill, London NW7 1AA, England); Mautner, V. *J Virol* 116(1): 19-24; 1976.

In an attempt to quantify the type-specific antibodies of adenovirus type 2 and 5 (Ad2 and Ad5), the radioimmunoprecipitation (RIP) method was used as a sensitive assay for the low levels of type-specific antibody that remain after all the cross-reacting antibody has been blocked with heterologous antigen. The virus neutralization titer of the antiserum was also determined following passage of the antiserum through immunoabsorbent columns of types 2 and 5 hexon. Assay of anti-fiber sera against Ad2 and Ad5 fiber indicated that 80% or more of the antibody was type-specific; the reverse was true for hexon. Direct titrations and saturation and competition inhibition methods were used to determine whether all the anti-hexon antibodies were cross-reactive but bound with different affinities to homologous and heterologous antigen. In the direct assays there was no sign of different avidities for homologous and heterologous binding, and in the inhibition studies, a significant amount (0.1-1%) of type-specific antibody remained after all cross-reacting antibodies had been absorbed with excess heterologous hexon. During the secondary immune responses of normal mice to anti-hexon serum, the proportion of type-specific antibodies was as high as 10-20%; this was confirmed for both types 2 and 5 hexon. The immunoabsorbent column experiments confirmed that the type- and group-specific antibodies were separate populations, since cross-reacting antibody could be removed without affecting the virus neutralization titer of the serum.

2686 ANTIGENIC DETERMINANTS OF ADENOVIRUS CAPSIDS. II. HOMOGENEITY OF HEXONS, AND ACCESSIBILITY OF THEIR DETERMINANTS, IN THE VIRION.

(Eng.) Willcox, N. (Natl. Inst. Medical Res., Mill Hill, London NW7 1AA, England); Mautner, V. *J Virol* 116(1): 25-29; 1976.

A sensitive radioimmunoprecipitation (RIP) technique for measuring type- and group-specific antibodies was used to study the accessibility of the corresponding antigenic determinants on the adenovirus virion. The capacity of purified peripentonal hexons and nonamers to bind each kind of antibody was also investigated. Adenovirus 5 (Ad5) virus was used to inhibit the reaction between [^{125}I]Ad5 hexon and antiserum to Ad2 hexon. The virions bound type-specific and cross-reactive antibodies impartially and much less effectively than free hexon or disrupted virus. Type-specific and cross-reactive determinants in Ad5 nonamers were assayed by their ability to detect or absorb the corresponding antibodies, or by the antibody response they elicited themselves. The results indicated that the nonamers possessed both type-specific and cross-reactive determinants, although some of the latter were less accessible in nonamers than in free hexon. Furthermore, Ad2 and Ad5 peripentonal hexons also possessed both kinds of determinants and in approximately the same proportions as in free hexons. The data argue strongly against the existence of a subset of hexons, especially rich in type-specific determinants, which are targets for neutralizing antibody. It is concluded that the mechanism of neutralization by antibody is complicated, and that the type-specific determinants exposed on the virion play a crucial role.

2687 INTERACTIONS OF KB-CELL GLYCOPROTEINS WITH AN ADENOVIRUS CAPSID PROTEIN.

(Eng.) Meager, A. (Natl. Inst. Medical Res., Mill Hill, London NW7 1AA, England); Butters, T. D.; Mautner, V.; Hughes, R. C. *Eur J Biochem* 61(2): 345-353; 1976.

The interactions of water-soluble glycoprotein(s) from KB cells with adenovirus type 5 (Ad5) fiber protein were studied. ^{35}S- and ^{3}H-labeled, Ad5-infected KB cells were treated with trichlorotrifluoroethane, and the water-soluble extract components were purified by DEAE-Sephadex A50 ion-exchange chromatography and affinity chromatography on a fiber-Sepharose support. The elution profile of the [^{3}H]glycoproteins from the DEAE-Sephadex column closely followed that of the ^{35}S-labeled viral components. However, about equal amounts of ^{3}H-radioactivity were eluted in pools I (hexon region) and II (fiber-penton region), whereas considerably more ^{35}S was associated with pool II than with pool I. Dodecylsulfate-polyacrylamide gel electrophoresis indicated that the two fiber fractions obtained by DEAE-Sephadex chromatography were of host cell origin. The profile of ^{3}H was not, however, changed substantially after trypsinization. It appeared unlikely that the ^{3}H-labeled glycoproteins formed any part of the fiber structure, and fractionation of uninfected KB cells suggested that specific binding of KB glycoproteins to fiber was probably not a prerequisite for their copurification with fiber on DEAE-Sephadex. When extracts of uninfected ^{3}H-labeled KB cells were subjected to fiber-Sepharose affinity chromatography, about 5% of the total radioactivity was retained; this was increased to 25% after DEAE-Sephadex chromatography of the extract and to 77% after trypsinization. The absorbed radioactivity contained predominantly high molecular wt glycoproteins, with some smaller heterogeneous glycoproteins. The glycoproteins specifically binding to fiber appeared to be carbohydrate-rich, particularly after trypsinization.

2688 ANALYSIS OF MULTIPLE VIRAL GENOME FRAGMENTS IN ADENOVIRUS 7-TRANSFORMED HAMSTER CELLS.

(Eng.) Fujinaga, K.; Sekikawa, K.; Yamazaki, H.; Green, M. In: *Tumor Viruses. Cold Spring Harbor Symposia on Quantitative Biology.* Cold Spring Harbor Lab. (Cold Spring Harbor, NY): vol. 39, pp. 633-636; 1975.

In characterization experiments with viral DNA, the presence of 200-400 copies of around 10%-20% of the viral genome was detected in adenovirus 7-transformed cells. A new method based on the kinetic analysis of labeled viral DNA reassociation in the presence of unlabeled transformed cell DNA was employed. DNA-DNA homology measurement utilizing defined DNA fragments (restriction endonuclease digests) as labeled probes supported the above conclusion. This procedure is of general value in analyzing the number and fraction of viral gene sequences in cells transformed by DNA and RNA tumor viruses.

2689 TRANSFER OF DNA FROM ADENOVIRUS INTO THE NUCLEUS AT THE BEGINNING OF THE INFECTION OF HELA CELLS. (Fre.) Ogier, G. (Unite de Virologie fondamentale et appliquee, 1, place du Pr J.-Renaut, 69008 Lyon, France); Chardonnet, Y.; Chantepie-Auray, J. *C R Acad Sci (D) (Paris)* 282(2): 247-250; 1976.

The transfer of adenovirus 7 (Ad7) DNA to HeLa cell nuclei and the possibility of modifying the transfer through viral inactivation or cell treatment were investigated. Ad5 (control) and Ad7 were labeled with ^{32}P and ^{3}H and placed in calf serum containing HeLa cells at either 4 C or 37 C. Other Ad7 were inactivated by UV radiation, and some HeLa cells were treated with chloroquine. The passage of Ad7 into the nucleus was the same as that of Ad5 although it began later. The rate of transfer was greater at 37 C than at 4 C. The DNA of the inactivated virus was taken up as efficiently as the normal virus; this may, however, be DNA from viruses that were unaffected. The chloroquine-treated cells took up DNA as well as the nontreated ones. Alteration of the lysosomes by chloroquine apparently did not affect the passage of viral DNA.

2690 CHARACTERIZATION OF AN ADENOVIRUS EARLY PROTEIN REQUIRED FOR VIRAL DNA REPLICATION: A SINGLE STRAND SPECIFIC DNA BINDING PROTEIN. (Eng.) Levine, A. J. (Princeton Univ. Dept. Biochemical Sciences, Princeton, NJ 08540); van der Vliet, P. C.; Rosenwirth, B.; Anderson, C.; Rabek, J.; Levinson, A.; Anderson, S. *Mol Cell Biochem* 11(2): 79-95; 1976.

An attempt was made to characterize the single-strand-specific DNA binding proteins of human adenovirus types 2, 5 and 12. Results indicated that the molecular wt of these proteins were 72,000 for types 2 and 5 and 60,000 for type 12. In all three cases proteolytic breakdown fragments of these binding proteins (molecular wt 48,000) were also observed. Analysis of the methionine containing tryptic peptides of these proteins indicated that the types 2 and 5 proteins were similar and clearly distinguishable from the type 12 protein. The peptide maps of these three viral proteins were clearly different from a similar protein found in mock infected cells. Temperature sensitive mutants of type 5 (H5ts125) and type 12 (H12tsA275) adenoviruses failed to produce these proteins at the nonpermissive temperature. H5ts125 infected cells grown at the permissive temperature produced a protein (molecular wt 72,000) that was thermolabile for continued binding to DNA, when compared to type 5 wild type adenovirus protein (molecular wt 72,000). An analysis of the phenotype of this adenovirus mutant indicated that it coded for a viral function early in the infection that was required for viral DNA replication.

2691 RECENT STUDIES ON THE CHARACTERISTICS OF ADENOVIRUS-INFECTED AND -TRANSFORMED CELLS. (Eng.) McDougall, J. K.; Dunn, A. R; Gallimore, P. H. In: *Tumor Viruses. Cold Spring Harbor Symposia on Quantitative Biology*. Cold Spring Harbor Lab. (Cold Spring Harbor, NY): vol. 39, pp. 591-599; 1975.

The antigenic characteristics and oncogenicity of rat cells and mouse/human hybrid cells infected and transformed with adenovirus (Ad) types 2 and 12 were studied. Cells arrested in metaphase were studied by Giemsa banding, and cells treated with AD2 fiber, hexon, or T antigen antisera were studied by indirect immunofluorescence. In situ hybridization experiments were also carried out with Ad cRNA. Rat embryo cells from different tissues derived from three strains of rat proved semipermissive for Ad2; at 72 hr after infection 38% of the cells were positive for viral DNA synthesis, and the virus yield per cell was 1 to 2 x 10^3 PFU/cell. There was a close correlation between transformation efficiency and the initial levels of viral antigen synthesis. The cells appeared to contain only incomplete viral genomes. The initiation of tumor development in syngeneic rats depended on the suppression of immune response, but the transplantability of the tumor was not dependent on immunosuppression. Characteristics of the Ad2- and Ad12-transformed rat cells are reported. The transformed cell lines contained a high percentage of new marker chromosomes, and many cells showed rings, dicentrics, gaps, breaks, and fragments. Similar changes were seen in Ad12-transformed human cells. Specific chromosome aberrations and gene localization are being investigated with mouse/human hybrid cells infected with Ad12. The results suggest that tolerance or rejection of an Ad-transformed cell might depend on the virus-induced expression of cellular genes. The location of the viral genes within the host genome may be of prime importance. (40 references)

2692 NUCLEIC ACID RENATURATION AND RESTRICTION ENDONUCLEASE CLEAVAGE ANALYSES SHOW THAT THE DNAs OF A TRANSFORMING AND A NONTRANSFORMING STRAIN OF EPSTEIN-BARR VIRUS SHARE APPROXIMATELY 90% OF THEIR NUCLEOTIDE SEQUENCES. (Eng.) Sugden, B. (McArdle Lab., Univ. Wisconsin, Madison, WI 53706); Summers, W. C.; Klein, G. *J Virol* 18(2): 765-775; 1976.

Viral DNA molecules were purified from a nontransforming and a transforming strain of Epstein-Barr virus (EBV) by the EcoRl restriction endonuclease digestion. Each viral DNA was labeled, by use of avian myeloblastosis virus DNA polymerase, in vitro and renatured in the presence of an excess of either one or the other unlabeled viral DNA. The results of these experiments support three general conclusions: (a) the DNAs of these two strains of Epstein-Barr virus share approximately 90% of their nucleotide sequences; (b) both viral DNA populations are reasonably homogeneous; and (c) both DNAs contain repetitions or inverted repetitions of some of their nucleotide sequences.

2693 INHIBITION OF PRODUCTIVE REPLICATION OF EPSTEIN-BARR VIRUS DNA BY PHOSPHONOACETIC ACID. (Eng.) Yajima, Y. (Rush-Presbyterian-Saint Luke's and Univ. Illinois Medical Centers, Chicago, IL 60612); Tanaka, A.; Nonoyama, M. *Virology* 71(1): 352-354; 1976.

The effect of phosphonoacetic acid (PAA) on Epstein-Barr virus (EBV) DNA replication was studied in superinfected Raji cells. Nonproductive cells that contained 50 latent EBV genomes per cell were superinfected with an EBV concentrate and labeled with ^{3}H-thymidine 13-40 hr later. The superinfection completely inhibited host cell DNA synthesis, whereas viral DNA replicated extensively. Addition of PAA (100 μg/ml) at the time of superinfection caused complete suppression of viral DNA replication; DNA synthesis in uninfected cells was not affected. Synthesis of host DNA in superinfected cells was still completely inhibited even under the condition that suppressed viral DNA synthesis. When the cells were cultured in the presence of the same concentration of PAA for 3 wk, the number of EBV genomes per cell remained unchanged and the cells grew normally. The results indicate that productive replication of EBV DNA in this system is sensitive to the action of PAA, while replication of latent EBV DNA and cellular DNA is not. P3 HRI cells that contained 340 genome equivalents per cell were cultivated in the presence of the same concentration of PAA for 3 wk by diluting the culture tenfold each week. The percentage of cells positive for early antigen decreased from 10% before treatment to 3% after treatment. The number of genome equivalents decreased from 340 to 10 per cell following treatment.

2694 COVALENTLY CLOSED CIRCULAR DUPLEX DNA OF EPSTEIN-BARR VIRUS IN A HUMAN LYMPHOID CELL LINE. (Eng.) Lindahl, T. (Dept. Chemistry, Tumor Biology and Biochemistry, Darolinska Inst., 104 01 Stockholm, Sweden); Adams, A.; Bjursell, G.; Bornkamm, G. W.; Daschka-Dierich, C.; Jehn, U. *J Mol Biol* 102(3): 511-530; 1976.

A nonintegrating form of Epstein-Barr virus (EBV) DNA was purified from the Brukitt lymphoma-derived human lymphoid cell line Raji by CsCl density gradient centrifugation and neutral glycerol gradient centrifugation. This intracellular form of the virus DNA sedimented at a rate typical of a covalently closed circular DNA molecule of the size of the virus genome in both neutral and alkaline solution. X-irradiation (500 rads) led to a form with the sedimentation properties of open circular DNA (a circular duplex molecule containing one or more single-strand breaks). The direct observation of large circular DNA molecules by electron microscopy confirms the covalently closed circular duplex structure of part of the intracellular viral DNA. Such circular molecules were not detected in corresponding DNA fractions from EBV-negative human lymphoid cell lines. In ethidium bromide/CsCl density gradient centrifugation experiments, the purified nonintegrated virus DNA behaved as twisted, covalently closed DNA circles with two same initial superhelix density as polyoma virus DNA. With the latter technique intracellular EBV DNA was isolated in > 90% form from nonproducer cells. The molecular wt of the circular virus DNA from Raji cells, determined by countour length measurements, is the same (within experimental error) as that of the linear DNA from virus particles.

2695 EPSTEIN-BARR VIRUS-SPECIFIC RNA I. ANALYSIS OF VIRAL RNA IN CELLULAR EXTRACTS AND IN THE POLYRIBOSOMAL FRACTION OF PERMISSIVE AND NONPERMISSIVE LYMPHOBLASTOID CELL LINES. (Eng.) Hayward, S. D. (Dept. Medicine, Univ. Chicago, Il 60637); Kieff, E. D. *J Virol* 18(2): 518-525; 1976.

To determine the mechanisms involved in controlling Epstein-Barr (EBV) genome expression, viral RNA in permissive and nonpermissive EBV-infected lymphoblastoid cell lines (Raji, Namalwa, Kurgan, and HR-1 cultures) was analyzed by observation of the hybridization kinetics of labeled EBV HR-1 DNA with unlabeled RNA extracted from whole cells or from the polyribosomal fraction. RNA, homologous to only 3% of the EBV HR-1 DNA, was present in the polyribosomal fraction of the nonpermissive Namalwa and Kurgans cells; this suggests that function of only a small fraction of the EBV genome is required to express the EBV-related intranuclear antigen and to maintain lymphoblastoid cells in a transformed state. In general, the extent of the viral DNA sequences transcribed into stable RNA correlated with the extent of phenotypic expression of the EBV genome. RNA extracted from virus-producing HR-1 cells contained RNA sequences transcribed from at least 45% of the viral DNA, whereas the nonpermissive cell lines contained transcripts homologous to a much smaller proportion of the EBV DNA. Viral RNA sequences found in the polyribosomal fraction of HR-1 cells arose from almost the same template as the viral RNA sequences in extracts of unfractionated HR-1 cells. In contrast, in nonpermissive lymphoblastoid cells, less than 30% of the viral RNA species found in whole-cell extracts were identified in the polyribosomal fraction. These observations indicate that expression of EBV genetic information is regulated in at least two ways: first, by some mechanism that regulates which DNA sequences give rise to stable RNA; second, by a mechanism whereby certain viral RNA transcripts are selectively excluded from stable association with the polyribosomes.

2696 COMPARISON OF THE SYNTHESIS OF THE POLYOMA S ANTIGEN AND THE EPSTEIN-BARR VIRUS (EBV)-ASSOCIATED MEMBRANE ANTIGEN (MA). (Eng.) Ernberg, I. (Dept. Tumor Biology, Karolinska Institutet, 104 01 Stockholm 60, Sweden); Klein, G.; Birg, F.; Meyer, G. *Cancer Biochem Biophys* 1(3): 111-114; 1975.

The synthesis of polyoma surface (S) antigen during abortive transformation is contrasted with that of Epstein-Barr virus (EBV)-associated membrane antigen (MA). Although during this period a virally determined transient MA is induced to appear on the majority of target cells in both cases, there are some significant differences. Unlike polyoma, EBV specifies cell membrane antigens that are also present on the virus particle, appearing on the outside envelope. Whereas inhibitors of DNA synthesis, such as cytosine arabinoside, bromodeoxyuridine, or mitomycin C, prevent the synthesis of the polyoma S antigen, they have no such effect on the EBV-induced membrane antigen. Actinomycin D inhibits synthesis of MA but not of polyoma S. The main difference between the polyoma and the EBV system is the dependence of the membrane antigen synthesis on at least one cycle of DNA synthesis in the case of polyoma and its apparently complete independence in the case of EBV. This might be due to the fact that polyoma must integrate with the cell genome before it can utilize the cellular apparatus for transcription and translation, while EBV can initiate and carry out this process.

2697 AN EBV-GENOME-NEGATIVE CELL LINE ESTABLISHED FROM AN AMERICAN BURKITT LYMPHOMA; RECEPTOR CHARACTERISTICS. EBV INFECTIBILITY AND PERMANENT CONVERSION INTO EBV-POSITIVE SUBLINES BY IN VITRO INFECTION. (Eng.) Klein, G. (Dept. Tumor Biology, Karolinska Institutet, S-104 01 Stockholm, 60, Sweden); Giovanella, B.; Westman, A.; Stehlin, J. S.; Mumford, D. *Intervirology* 5(6): 319-334; 1975.

An in vitro line was derived from the Burkitt lymphoma (designated Ra No. 1) of a 3-yr-old boy, that produced malignant tumors when inoculated into thymus-deficient nude mice. The cells had B-lymphocyte characteristics, with surface-associated μ and κ chains and Epstein-Barr virus (EBV) receptors, and were readily infected with EBV in vitro. B95-8 virus induced EBV-determined nuclear antigen (EBNA) but not early antigen (EA) in Ra No. 1 cells, whereas P3HR-1 virus induced both EBNA and EA; however, the EA level was much lower than in the prototype Raji strain. EBNA and EA levels were comparable in Ra No. 1 and in the previously established EBV-infection-sensitive BJA-B lymphoma. The two lines differed, however, because BJA-B could be converted into a permanent EBV-carrying line by the B95-8 but not by the P3HR-1 virus strain, whereas Ra No. 1 could be converted by the P3HR-1 virus. This difference demonstrates that different B-lymphocyte-derived lymphoma lines can vary in the restrictive control they can exert on the same virus strain. Furthermore, virus strains vary in their sensitivity to restrictions of the same host cell. Permanent EBV conversion of the Ra No. 1 cell did not appear to change the cellular controls, as judged by the unchanged sensitivity of the cell to viral antigen (EA, viral capsid antigen) induction by P3HR-1 virus superinfection.

2698 EPSTEIN-BARR VIRUS (EBV)-INDUCED CHANGE IN THE SATURATION SENSITIVITY AND SERUM DEPENDENCE OF ESTABLISHED, EBV-NEGATIVE LYMPHOMA LINES IN VITRO. (Eng.) Steinitz, M. (Dept. Tumor Biology, Karolinska Inst., S-104 01 Stockholm 60, Sweden); Klein, G. *Virology* 70(2): 570-573; 1976.

The Epstein-Barr virus (EBV)-induced change in saturation sensitivity and serum dependence of an EBV-negative lymphoma line, Ra 1, was compared with four different EBV-converted Ra 1 sublines. In contrast to Ra 1, which died within less than 3 days after reaching maximal cell number per milliliter, the four converted lines maintained a high number of living cells per milliliter for a long time after they reached saturation density. The EBV-negative line appeared to be more sensitive to depletion of the medium (RPMI-1640) than the EBV-positive lines. The EBV-negative line may also have been more sensitive to the accumulation of growth inhibitory or toxic products in the used media. The EBV-negative line was unable to grow at low (0.75%) fetal calf serum levels, whereas all four converted lines grew well at this concentration.

2699 SYNTHESIS OF PROTEINS IN CELLS INFECTED WITH HERPESVIRUS. XI. SULFATED, STRUCTURAL GLYCOPROTEINS. (Eng.) Kaplan, A. S. (Vanderbilt Univ. Sch. Medicine, Nashville, TN 37232); Ben-Porat, T. *Virology* 70(2): 561-563; 1976.

Experiments were performed to determine whether or not structural glycoproteins of pseudorabies virus-infected monolayers of rabbit kidney cells are sulfated. The infected rabbit kidney cells were incubated with $^{35}SO_4^{2-}$ and [^{3}H]glucosamine, and 12 hr postinfection the culture fluids containing labeled virus were collected and analyzed on a linear sucrose gradient. Although the ratio of $^{35}S/^{3}H$ incorporated into nonstructural proteins, which are also synthesized by these cells, was about 6-10 times greater than that incorporated into the structural viral glycoproteins, the amount incorporated in the latter was significant. Sodium dodecyl sulfate-polyacrylamide gel electrophoresis confirmed that the small amount of $^{35}SO_4^{2-}$ associated with the virus peak was not due to contamination with 3a sulfated glycoprotein. Only the viral glycoproteins were sulfated (the other structural viral proteins were not); the function of the sulfate in these compounds is unknown.

2700 THE ROLE OF *HERPESVIRUS SAIMIRI* GENOMES IN ONCOGENIC TRANSFORMATION OF PRIMATE CELLS. (Eng.) Fleckstein, B. (Institut fuer klinische Virologie der Universitaet Erlangen-Nuernberg, Loschgestrasse 7, D-852 Erlangen, W. Germany); Bornkamm, G. W.; Werner, F. J. *Bibl Haematol (Basel)* (43): 308-312; 1976.

Herpesvirus saimiri M-DNA, one of two types of DNA molecules isolated from *H. saimiri*, was injected (50 ng to 6 μg) into 12 cotton-topped marmosets iv, im, or ip. One animal receiving 1 μg developed a typical *H. saimiri*-induced lymphoma, and the infectious virus was reisolated from the spleen cells by cocultivation with permissive owl monkey kidney cells. With the use of cell line 70 BM2 from the leukocytes of a leukemic marmoset with *H. saimiri* lymphoma (these cells do not produce infectious virus when cocultivated with permissive cells), the reassociation of radioactive L-DNA with 500 μg/ml of 70 BM2 DNA was compared with the reassociation rate in the presence of 500 ng/ml of cold purified L-DNA. Repetitive H sequences were found in a relative excess compared to L sequences with regard to the ratio of H- to L-DNA in the M genomes of *H. saimiri* virions. More than 80% of the radioactive L-DNA and more than 88% of radioactive H-DNA were driven into hybrids by 70 BM2 DNA. It is concluded that most or even all of the *H. saimiri* DNA sequences are present in the 70 BM2 nonproducer cell line. Autoradiography after hybridization of 70 BM2 cell smears with H-specific highly labeled cRNA suggested an association of viral DNA sequences with cellular chromosomes. Significant hybridization of L-DNA with RNA from two non-virus-producing lines (1670 and 70 N2) and from two tumor biopsies indicated that L-DNA was transcribed considerably in transformed cells. (9 references)

2701 PSEUDOTYPE FORMATION BETWEEN HERPES SIMPLEX VIRUS AND RNA VIRUSES. (Eng.) Huang, A. S. (Harvard Medical Sch., Boston, MA 02115); Hewlett, N. *Bibl Haematol (Basel)* (43): 313-317; 1976.

Data relating to the characteristics of pseudotypes containing vesicular stomatitis virus (VSV) genomes and herpes simplex virus (HSV) envelopes are presented, and attempts to produce HSV (VSV) and HSV (murine leukemia virus) pseudotypes are described. Cytosine arabinoside (20 μg/ml) was incorporated into the culture media, since it selectively inhibits HSV progeny during coinfection of HEp-2 cells with HSV and VSV. At different times after infection with HSV, cells were superinfected with VSV, at a multiplicity of 0.1 or 50. At 30 hr after the initiation of infection by HSV, extracellular virus containing only VSV genomes was harvested. Up to 30% of the progeny were resistant to anti-VSV serum and were presumably VSV(HSV) pseudotypes. The yield of pseudotypes was higher if cells were superinfected with VSV as soon after HSV as possible. Higher multiplicities of VSV also increased the yield of pseudotypes. VSV(HSV) pseudotypes did not initiate plaques on Chinese hamster ovary cells, and therefore they have a host range similar to HSV. VSV(HSV) were also susceptible to neutralization by anti-HSV antiserum. Attempts to produce HSV(VSV) and HSV(MuLV) were unsuccessful. The potential of these pseudotypes as laboratory tools is discussed. (14 references)

2702 HERPES TYPE-2 VIRUS ANTIGENS IN HUMAN CERVICAL CARCINOMA. (Eng.) Adelusi, B. (Dept. Obstretrics & Gynaecology, Univ. Coll. Hosp., Ibadan, Nigeria); Osunkoya, B. O.; Rabiyi, A. *Obstet Gynecol* 47(5): 545-548; 1976.

Exfoliated cells from the cervices of 22 patients with histologically diagnosed squamous cell carcinoma of the cervix and from those of 24 control women were examined for herpes subtype 2 (HT 2) antigens by indirect immunofluorescence. These women were seen in a clinic in Ibadan, Nigeria. Cells from all the patients with carcinoma showed fluorescence when stained with rabbit antiserum against HT-2 antigen. In contrast, squamous cells from the cervices of 24 control women with no cervical disease and of 2 women with chronic cervicitis did not contain such HT-2 antigens. These findings add to the evidence for an association between HT-2 and carcinoma of the cervix.

2703 UTERINE LEIOMYOSARCOMA IN THE RABBIT FOLLOWING IN SITU INOCULATION OF NUCLEIC ACID EXTRACT FROM CELL CULTURES INFECTED WITH HERPES SIMPLEX VIRUS TYPE 2 (HSV-2). (Eng.) Nastac, E. ("Stefan S. Nicolau" Inst. Virology, 285, Sos. Mihai Bravu, Bucharest, Romania); Athanasiu, P.; Stoian, M.; Hozoc, M.; Morfei, A. *Rev Roum Med Virol* 26(2): 149-150; 1975.

Six rabbits were inoculated with extracts of herpes simplex virus type 2-infected human embryo cell cultures via laparotomy into the uterine cervix. One rabbit developed a uterine leiomyosarcoma and died 36 days after inoculation. Of the remaining five, one was sacrificed, and positive immunofluorescence was demonstrated in the neurons at the base of the brain in the presence of specific herpes antiserum. Complement fixation antibodies to the virus were detected in four of the five rabbits with titers of 1/32-1/1,024.

2704 QUANTITATIVE CYTOPHOTOMETRY EVALUATED AS A METHOD FOR ANALYSES OF HERPES SIMPLEX VIRAL DEOXYRIBONUCLEIC ACID SYNTHESIS. (Eng.) Trusal, L. R. (Dept. Biology, Pennsylvania State Univ., University Park, PA 16802); Anthony, A.; Docherty, J. J. *Infect Immun* 13(5): 1503-1509; 1976.

Feulgen deoxyribonucleic acid (F-DNA) microspectrophotometry was evaluated as a potential tool to quantitate herpes simplex virus type 1 (HSV-1) and 2 (HSV-2) DNA synthesis in single human embryonic lung cells. Microspectrophotometric measures were correlated with biochemical data obtained with tritium-labeled thymidine (^{3}H-TdR). It was established that: (a) viral-induced increase in F-DNA can be cytophotometrically detected between 4 and 6 hr postinfection (pi), which corresponds to the initial incorporation of ^{3}H-TdR into viral DNA; (b) peak F-DNA levels occurred 8 hr pi, which corresponds to the time when there is a maximal uptake of ^{3}HTdR into viral DNA; (c) HSV-2-infected nuclei exhibited a faster initial (between 4 and 6 hr pi) elevation of F-DNA than HSV-1-infected nuclei (between 6 and 8 hr pi), which supported cytological observations of a more rapid development of an inclusion body in HSV-2-infected nuclei. Although F-DNA cytophotometry could not distinguish between cell and viral DNA, the overall study supports the existence of a good correlation between data obtained with the use of microspectrophotometric and conventional isotope methods. Furthermore, cytophotometry complemented biochemical evaluations in that it permitted analyses of DNA changes on a single-cell basis or the detection of infection in very small numbers of cells.

2705 REPRODUCTION OF TYPE 1 AND 2 HERPES SIMPLEX VIRUS STRAINS IN CELLS ADAPTED OR NOT ADAPTED TO 40 C. (Rus.) Szanto, J. (Inst. Virology, Slovakian Acad. Sciences, 809 39 Bratislava, Czechoslovakia). *Acta Virol (Praha)* 19(4): 287-292; 1975.

Eight strains of herpes simplex virus (HSV) type 1 and 6 strains of HSV type 2 were cultured in cells of rabbit lung that were adapted and not adapted to a temperature of 40 C. Marked cell-dependent differences were found in the ability of herpes viruses to replicate at raised temperatures. Adapted cells were the decisive system for some viral strains of both types in cultures at a supraoptimal temperature. Four of the HSV-2 strains reproduced relatively well in cells adapted to 40 C, compared with control cells cultured at 36 C. Although some type 1 strains reproduced poorly at 40 C, the infectious virus persisted in adapted cells cultured at 40 C longer than in those cultured at 36 C. Nonadapted cells of the rabbit lung were an indecisive system for some HSV strains of both types. HSV-2 did not reproduce in nonadapted cells at 40 C, while type 1 strains did. (9 references)

2706 ACTIVATION OF AN ENDOGENOUS MOUSE TYPE C VIRUS BY ULTRAVIOLET-IRRADIATED HERPES SIMPLEX VIRUS TYPES 1 AND 2. (Eng.) Hampar, B. (Natl. Cancer Inst., Natl. Insts. Health, Bethesda, MD 20014); Aaronson, S. A.; Derge, J. G.; Chakrabarty, M.; Showalter, S. D.; Dunn, C. Y. *Proc Natl Acad Sci USA* 73(2): 646-650; 1976.

Infection of BALB/c mouse cells with UV-irradiated herpes simplex virus (HSV) types 1 and 2 activated a xenotropic type C virus. The levels of type C virus activated were related to the UV dose and the multiplicity of infections. The ability of HSV to activate type C virus was eliminated by heat inactivation and by neutralization with specific antiserum against HSV, but was not affected by purification or treatment with DNase and RNase. Maximum levels of type C virus in the cells and medium were observed within 1 day with HSV infection, and the levels returned to control cell values within 3-4 days. These results raise the question of whether the putative oncogenic potential of HSV may involve alteration of the expression of type C virus information. Furthermore, the activation of the type C virus by HSV suggests that HSV is a cofactor in oncogenesis.

2707 TRANSFORMATION OF CELLS BY HERPES SIMPLEX VIRUS--FACT OR FANTASY? (Eng.) McAuslan, B. R.; Garfinkle, B.; Adler, R.; Devinney, D.; Florkiewicz, R.; Shaw, J. E. In: *Tumor Viruses. Cold Spring Harbor Symposia on Quantitative Biology*. Cold Spring Harbor Lab. (Cold Spring Harbor, NY): vol. 39, pp. 765-772; 1975.

A line of cells was studied to determine its response to herpes simplex virus (HSV) exposure. The replication of HSV types 1 and 2 is defective in XC cells, a continuous line of Wistar rat-derived cells that carry Rous sarcoma virus information. The resistance of these cells to the toxic effects of HSV was apparently not due directly to the presence of RNA tumor virus in these cells. Other lines transformed by RNA tumor viruses exhibited no comparable resistance. The nature of the association between HSV and the transformed cells for a number of generations is not yet established; on prolonged subculture, much of the original HSV information failed to be expressed. Not more than approximately 0.1 viral genome per cell remained permanently associated with the host DNA. The ''transformants'' had a number of characteristics that distinguished them from parental cells, and such characteristics in the light of the immunological data suggested that XC cells exposed to HSV could permanently retain HSV-specific information.

2708 INHIBITION OF INITIATION OF PROTEIN SYNTHESIS BY 7-METHYLGUANOSINE-5'-MONOPHOSPHATE. (Eng.) Hickey, E. D. (Dept. Biological Sciences, State Univ. New York, Albany, NY 12222); Weber, L. A.; Baglioni, C. *Proc Natl Acad Sci USA* 73(1): 19-23; 1976.

The functional role for the 7-methylguanosine-5'-monophosphate ($m^7G^{5'}p$) nucleotide sequence in mRNA translation was studied by translation of rabbit globin mRNA and other mRNAs in the presence of mononucleotide analogs of the 5'-terminal end of this mRNA. Translation was studied in wheat germ extract and in a different cell-free system. Translation of the rabbit globin mRNA in the wheat germ protein-synthesizing system was inhibited by $m^7G^{5'}p$ but not by other guanosine nucleotides without the 7-methyl group or with the phosphate in a different position. The translation of the mRNA of tobacco mosaic virus and poly(A) + HeLa mRNA was also inhibited by $m^7G^{5'}p$. Study of the formation of initiation complexes between reovirus RNA labeled in the methyl group of the 5'-terminal guanosine and wheat germ ribosomes demonstrated that $m^7G^{5'}p$ acts at the level of initiation of protein synthesis. $m^7G^{5'}p$ did not inhibit the translation of poly(U) and RNA of satellite tobacco necrosis virus, which do not have the 5'-terminal sequence $m^7G^{5'}ppp$. In the case of the satellite tobacco necrosis virus RNA, however, some stimulation of its translation was consistently observed in the presence of $m^7G^{5'}p$. It is suggested that $m^7G^{5'}p$ interacts with a site on initiation factor(s) or ribosomal subunits that is involved in mRNA recognition, presumably by binding to the 5'-terminal sequence of $m^7G^{5'}ppp$.

2709 XC-CELL FUSION INDUCED BY MURINE PLASMOCYTOMA CELLS. III. RNA GENE EXPRESSION CORRELATED TO SYNCYTIUM FORMATION. (Eng.) Herisse, J. (Unite de Recherche de Virologie no. 102 de l'I.N.S.E.R.M., 2, place de Verdun, 59045 Lille Cedex, France); Lemay, P.; Tavitian, A. *Biomedicine* 25(1): 3-7; 1976.

The viral gene expression in MF_2 cells (a mouse myeloma derived cell line) was studied both in vivo and in vitro. A DNA complementary to viral RNA (cDNA) was synthesized by in vitro transcription of the 70S RNA using the RNA-directed DNA polymerase (reverse transcriptase endogenous activity) associated with the viral particles. This cDNA was used as a probe to detect virus-specific nucleic acid sequences in the poly A-RNA extracted from the MF_2 cells maintained in various culture conditions, using hybridization techniques. The cDNA was hybridized more readily and efficiently to the Poly A-rich RNA extracted from in vitro cultured MF_2 cells than from the same RNA from the in vivo ones. As shown by molecular hybridization, the DNA complementary to the viral 70S RNA anneals to the in vitro cultured cells at a higher rate than it does to the homologous RNA fraction from ascitic cells, suggesting that there are more viral sequences in the former than in the latter. This evidence supports the hypothesis that the cytopathogenic effect is linked to the expression of budding C type particles.

2710 RECENT DATA ON PROGRESSIVE MULTIFOCAL LEUKOENCEPHALOPATHY. ONCOGENIC ROLE OF PAPOVA VIRUS IN MAN? (Fre.) Castaigne, P. (Cliniques des Maladies du Systeme nerveux, Hopital de la Salpetriere, 47, boulevard de l'Hopital, 75634 Paris Cedex 13, France). *Acad Nat Med* 159(7): 660-664; 1975.

The association of the simian virus 40 (SV40) papovavirus and multifocal, progressive leukoencephalopathy (MPL) is discussed and confirmed in a report of five cases. Diagnosis on the basis of clinical symptoms, i.e., cortical blindness and mental confusion, was made in two cases, one of which was confirmed by cerebral biopsy. In the other cases, diagnosis was made by anatomical examination. Typically, the biopsy or autopsy specimens of the brain showed poorly defined areas of demyelinization of variable size, more or less confluent, disseminated in the subcortical white matter, occasionally encroaching on the grey cortex. Examination by the electron microscope revealed modification of the oligodendroglia cells and giant astrocytes, often multinucleated. In all cases, intranuclear inclusions, morphologically identical to the SV40 virus, were present in the oligodendroglia. The nuclei were homogeneous in these cells, and the chromatin was concentrated in a border around the nuclear membrane. One case, a child with hemolytic anemia and a spinal-ganglionic syndrome with hypergammaglobulinemia and total immunological defect, had tumor nodules adjacent to the MPL lesions. At autopsy, the nodules were identified as gliomas. An oncogenic role of the SV40 papovavirus is proposed in this case, although an antigen to the virus was not found in the cerebral parenchyma nor were virus particles found in the tumor tissue. An absence of immune defenses in the child may have played a role in the unusual tumor-MPL pathology. (12 references)

2711 IMMUNOFLUORESCENCE ON FIXED CELLS FOR VIRAL ANTIGENS. (Eng.) Hilgers, J. (Netherlands Cancer Inst., Amsterdam, The Netherlands). *Int Union Cancer Tech Rep Ser* 20: 121-123; 1975.

Techniques for indirect immunofluorescence and immunofluorescence absorption to determine the presence and the intracellular deposition of viral antigens in infected cells are described. The indirect immunofluorescence test can be done on acetone-fixed cells. Immunofluorescence absorption tests can be done on lyophilized tumor, tissue extracts, or with 20% tissue and tumor homogenates in saline. The immunofluorescence absorption test is of the same sensitivity as the complement-fixation test and about 1000 times less sensitive than radioimmunoassay. Antisera against viruses often contain antibodies to normal cellular components. These can be absorbed in vitro with virus-negative tissue extracts or in vivo in the species of origin of the virus.

2712 ELECTRON MICROSCOPIC STUDY OF VIRUS PARTICLES IN YOLK SAC AND PLACENTA AND IN HEMATOPOIETIC ORGANS OF NEWBORN C3HF MICE. (Eng.) Feldman, D. (Dept. Experimental Pathology, Hoffmann-La Roche, Inc., Nutley, NJ 07110); Gross, L.; Swarm, R. L. *Cancer Res* 36(5): 1814-1820; 1976.

Virus particles in mouse yolk sac, placenta, and hematopoietic organs of newborn mice (1.5-18 days old) were studied by electron microscopy, and the relative number of C-type virus particles present in blood-forming organs from embryonic, newborn and adult C3Hf mice were compared. Yolk sac from a C3Hf and a C3H mouse contained C-type virus particles in the blood islands. Particles were observed budding from the plasma membrane of hemocytoblasts, from erythroblasts, and occasionally from reticulocytes. C-type particles were also found in similar cells in hematopoietic foci in the liver, spleen, and bone marrow of embryos, and they continued to be present in newborn C3Hf mice up to 11 days of age. Particles consistently appeared in the thymus, even in older suckling mice. In newborn C3Hf mice, the particles were observed in 8 of 9 thymus glands as compared to 8 of 8 in the embryos and 6 of 9 in the adults. In spleen, liver, and bone marrow, the decrease in the number of particles in older mice was more pronounced. C-type particles were not observed in the chorioallantoic placentas from mice that were given injections of mouse leukemia virus (Gross) or from normal noninjected mice; however, intracisternal A-type particles were present in cytotrophoblast cells from these placentas. The results indicate an association between C-type virus particle production and hematopoiesis in C3Hf mice.

2713 INFECTIOUS DNA FROM CELLS INFECTED WITH ROUS SARCOMA VIRUS, RETICULOENDOTHELIOSIS VIRUS OR ROUS-ASSOCIATED VIRUS-0. (Eng.) Cooper, G. M.; Temin, H. M. In: *Tumor Viruses. Cold Spring Harbor Symposia on Quantitative Biology*. Cold Spring Harbor Lab. (Cold Spring Harbor, NY): vol. 39, pp. 1027-1032; 1975.

2714 HOMOLOGY BETWEEN AVIAN ONCORNAVIRUS RNAs AND DNA FROM SEVERAL AVIAN SPECIES. (Eng.) Shoyab, M. (Univ. California Sch. Medicine, Los Angeles, Calif. 90024); Baluda*, M. A. *J Virol* 16(6): 1492-1502; 1975.

2715 FINE STRUCTURAL STUDY OF ASSOCIATED HERPESVIRUS: HERPESVIRUS OF TURKEY (HVT-FC126) (Proceedings Abstract). (Fre.) Tektoff, J. (Iffa-Merieux, Service de Microscopie Electronique, 254, rue M.-Merieux, 69007 Lyon, France); Gaudry, D.; Durafour, M. *J Microsc Biol Cell* 23(2): 81a; 1975.

2716 COMPARATIVE ASPECTS OF ETIOLOGY AND DIAGNOSIS. SOME CHARACTERISTICS OF THE CELL CULTURES ESTABLISHED FROM THE TUMOROUS TISSUES OF CATTLE WITH BOVINE LEUKOSIS. (Eng.) Takatori, I. (Natl. Inst. Animal Health, Kodaira, Tokyo 187, Japan); Yamamoto, H.; Yoshino, T.; Usui, T.; Mitsumori, K. *Bibl Haematol (Basel)* (43): 354-359; 1976.

2717 CELL POPULATIONS AND KNOWN SURFACE ANTIGENS OF TUMORS INDUCED BY ABELSON VIRUS IN PRISTANE-PRIMED BALB/C MICE: AN ANALYSIS BY IMMUNO-ELECTRON MICROSCOPY. (Eng.) Aoki, T. (Natl. Cancer Inst., Bethesda, Md. 20014); Potter, M.; Sturm, M. M.; Liu, M.; Walling, M. J. *J Natl Cancer Inst* 55(5): 1097-1106; 1975.

2718 IN VITRO TRANSMISSION OF MURINE LEUKEMIA VIRUS TO RESTRICTIVE MOUSE CELLS: NECESSITY OF MIXED INFECTION BY TWO DIFFERENT LEUKEMIA

VIRUSES. (Fre.) Yoshikura, H. (Fondation Curie, Institut du Radium, Section de Biologie, Batiment 110, 91400 Orsay, France). *C R Acad Sci (D) (Paris)* 281(14): 1063-1065; 1975.

2719 TRANSMISSION OF AUTO-IMMUNE HAEMOLYTIC ANAEMIA AND MURINE LEUKAEMIA VIRUS IN NZB-BALB/C HYBRID MICE. (Eng.) East, J. (Imperial Cancer Res. Fund, Buttonhole Lane, Mill Hill, London NW7 1AD, England); Harvey, J. J.; Tilly, R. J. *Clin Exp Immunol* 24(1): 196-209; 1976.

2720 IMMUNOFLUORESCENCE STUDIES ON THE ASSOCIATION BETWEEN A C-TYPE ONCORNAVIRUS AND RENAL GLOMERULOPATHY IN *PRAOMYS (MASTOMYS) NATALENSIS*. (Eng.) Van Pelt, F. G. (Inst. Experimental Gerontology TNO, Rijswijk ZH, The Netherlands); Bentvelzen, P.; Brinkhof, J.; Mannetje, A. H. 'T.; Zurcher, C. *Clin Immunol Immunopathol* 5(1): 105-118; 1976.

2721 INHERITED RESISTANCE TO N- AND B-TROPIC MURINE LEUKEMIA VIRUSES IN VITRO: TITRATION PATTERNS IN STRAINS SIM AND SIM.R CONGENIC AT THE FV-1 LOCUS. (Eng.) Schuh, V. (Dept. Anatomy, Univ. Toronto, Toronto, Canada, M5S 1A8); Blackstein, M. E,; Axelrad, A. A. *J Virol* 18(2): 473-480; 1976.

2722 A BIOCHEMICAL AND GENETIC ANALYSIS OF MAMMALIAN RNA-CONTAINING SARCOMA VIRUSES. (Eng.) Scolnick, E. M.; Goldberg, R. J.: Park, W. P. In: *Tumor Viruses. Cold Spring Harbor Symposia on Quantitative Biology.* Cold Spring Harbor Lab. (Cold Spring Harbor, NY): vol. 39, pp. 885-896; 1975.

2723 ENDOGENOUS PRIMATE AND FELINE TYPE C VIRUSES. (Eng.) Todaro, G. J.; Benveniste, R. E.; Callahan, R.; Lieber, M. M.; Sherr, C. J. In: *Tumor Viruses. Cold Spring Harbor Symposia on Quantitative Biology.* Cold Spring Harbor Lab. (Cold Spring Harbor, NY): vol. 39, pp. 1159-1168; 1975.

2724 SURFACE PROPERTIES OF C-TYPE VIRUS-INFECTED CELLS. (Eng.) Maruyama K.; Miller, M. F.; Hixson, D. C.; Wagner, S. H. In: *Cellular Membranes and Tumor Cell Behavior. A Collection of Papers Presented at the Twenty-Eighth Annual Symposium on Fundamental Cancer Research, 1975.* University of Texas System Cancer Center, M. D. Anderson Hospital and Tumor Institute. (Baltimore: Williams and Wilkins Co.): pp. 421-446; 1975.

2725 DETECTION OF VIRAL GENOMES BY HYBRIDIZATION IN SITU (Abstract). (Fre.) Langelier, Y. (Faculte de Medecine, Universite de Sherbrooke, Sherbrooke, Quebec, Canada). *J Microscop Biol Cellul* 23(2): 56a; 1975.

2726 TRANSFORMATION OF PRIMATE AND RODENT CELLS BY TEMPERATURE-SENSITIVE MUTANTS OF SV40. (Eng.) Butel, J. S.; Brugge, J. S.; Noonan, C. A. In: *Tumor Viruses. Cold Spring Harbor Symposia on Quantitative Biology.* Cold Spring Harbor Lab. (Cold Spring Harbor, NY): vol. 39, pp. 25-36; 1975.

2727 REACTIVATION OF SQUIRREL MONKEY HEART ISOLATE (HERPESVIRUS SAIMIRI) STRAIN FROM LATENTLY INFECTED HUMAN CELL CULTURES AND INDUCTION OF MALIGNANT LYMPHOMA IN MARMOSET MONKEYS. (Eng). Daniel, M. D. (Harvard Medical Sch., 1 Pine Hill Drive, Southborough, MA 01772); Silva, D.; Jackman, S. D.; Sehgal, P.; Baggs, R. B.; Hung, R. D.; King, N. W.; Melendez, L. V. *Bibl Haematol (Basel)* (43): 392-395; 1976.

2728 INTERPRETATION OF NUCLEAR LESIONS INDUCED BY HERPESVIRUS, SV40, AND RUBEOLA (Proceedings Abstract). (Fre.) Dupuy-Coin, A.-M. (Laboratoire de Pathologie Cellulaire, Centre des Cordeliers (Universite de Paris VI), 21, rue de l'Ecole-de-Medecine, 75270 Paris Cedex 06, France). *J Microsc Biol Cell* 23(2): 46a; 1975.

2729 FREQUENCY OF ANTIBODY TO A VIRUS-INDUCED TUMOR-ASSOCIATED ANTIGEN (AG-4) IN JAPANESE SERA FROM PATIENTS WITH CERVICAL CANCER AND CONTROLS. (Eng.) Kawana, T. (Dept. Obstetrics and Gynecology, Univ. Tokyo, Japan); Cornish, J. K.; Smith, M. F.; Aurelian, L. *Cancer Res* 36(6): 1910-1914; 1976.

2730 EXCRETION OF SPECIFIC GLYCOPROTEINS BY CELLS INFECTED WITH HERPES SIMPLEX VIRUS, TYPES 1 AND 2. (Eng.) Kaplan, A. S. (Vanderbilt Univ. Sch. Medicine, Nashville, TN); Erikson, J. S.; Ben-Porat, T. *Prog Med Virol* 21: 1-12; 1975.

2731 TRANSFORMATION OF EMBRYONIC RAT FIBROBLASTS BY HERPES SIMPLEX VIRUS TYPE 2. (Eng.) Munk, K.; Darai, G. In: *Oncogenesis and Herpesvirus II. Part 1: Biochemistry of Viral Replication and In Vitro Transformation. Proceedings of a Symposium held in Nuremburg, Federal Republic of Germany, 14-16 October 1974.* International Agency for Res. on Cancer. (Lyon, France): pp. 437-443; 1975.

2732 BLOCK TO MULTIPLICATION OF ADENOVIRUS SEROTYPE 2 IN MONKEY CELLS. (Eng.) Klessig, D. F. (Cold Spring Harbor Lab., Cold Spring Harbor, N.Y. 11724); Anderson, C. W. *J Virol* 16(6): 1650-1668; 1975.

2733 MAPPING OF ADENOVIRUS 2 RNA SEQUENCES IN LYTICALLY INFECTED CELLS AND TRANSFORMED CELL LINES. (Eng.) Sharp, P. A.; Gallimore, P. H.; Flint, S. J. In: *Tumor Viruses. Cold Spring Harbor Symposia on Quantitative Biology.* Cold Spring Harbor Lab. (Cold Spring Harbor, NY): vol. 39, pp. 457-474; 1975.

2734 THE VIRAL DNA REPLICATION MACHINERY OF ADENOVIRUS 12. (Eng.) Shimojo, H.; Shiroki, K.; Yamaguchi, K. In: *Tumor Viruses. Cold Spring*

Harbor Symposia on Quantitative Biology. Cold Spring Harbor Lab. (Cold Spring Harbor, NY): vol. 39, pp. 533-538; 1975.

2735 FINE STRUCTURE OF A TYPE B (BJA-B) CELL LINE DERIVED FROM BURKITT'S LYMPHOMA: NEGATIVE EBV GENOME (Proceedings Abstract). (Fre.) Garzon, S. (Faculte de Medecine, Universite de Montreal, C.P. 6128, Montreal 101, P.Q., Canada); Menezes, J.; Zollinger, M. *J Microsc Biol Cell* 23(2): 49a; 1975.

2736 FINE STRUCTURAL CHARACTERISTICS OF DIFFERENT LYMPHOBLAST LINES WITH THE GENOME OF EPSTEIN-BARR VIRUS (EBV) (Proceedings Abstract). (Fre.) Berthiaume, L. (Institut de Microbiologie et d'Hygiene de Montreal, 531, boulevard de Prairies, ville de Laval, P.Q. Canada, H7N 4Z3); Alain, R.; Joncas, J. H.; Menezes, J. *J Microsc Biol Cell* 23(2): 29a-30a; 1975.

2737 INTRACELLULAR DISTRIBUTION AND SEDIMENTATION PROPERTIES OF VIRUS-SPECIFIC RNA IN TWO CLONES OF BHK CELLS TRANSFORMED BY POLYOMA VIRUS. (Eng.) Maxwell, I. H. (Univ. Colorado Sch. Medicine, Denver, CO 80220). *J Virol* 18(2): 461-472; 1976.

2738 A SURVEY ON THE EFFECT OF STEROID HORMONE ON TYPE C VIRUS PRODUCTION FROM CULTURED MURINE CELLS. (Eng.) Wu, A. M. (Bionetics Res. Lab., Bethesda MD 20014); Richardson, L. S.; Paran, M.; Gallo, R. C. *Cancer Res* 36(6): 2025-2030; 1976.

2739 THE GENOME OF RNA TUMOR VIRUSES: A FUNCTIONAL REQUIREMENT FOR A POLYPLOID STRUCTURE? (Eng.) Cooper, P. D.; Wyke, J. A. In: *Tumor Viruses. Cold Spring Harbor Symposia on Quantitative Biology*. Cold Spring Harbor Lab. (Cold Spring Harbor, NY): vol. 39, pp. 997-1004; 1975.

2740 INTRACISTERNAL A PARTICLES FROM FLOPC-1 BALB/C MYELOMA: PRESENCE OF HIGH-MOLECULAR-WEIGHT RNA AND RNA-DEPENDENT DNA POLYMERASE. (Eng.) Kruegarger, R. G. (Coll. Medicine, Univ. Cincinnati, OH 45267). *J Virol* 18(2); 745-756; 1976.

2741 MORPHOLOGIC STUDY OF VIRUS-LIKE PARTICLES IN A CASE OF ACUTE LEUKEMIA. (Eng.) Foa, C. (Unite 119 de l'I.N.S.E.R.M. 27, Bd Lei Roure, 13009 Marseille, France); Foa, J.; Carcassonne, Y. *Cancer* 37(4): 1718-1724; 1976.

See also:

* (Rev): 2409, 2416, 2517, 2418, 2419, 2420, 2421, 2422, 2423, 2424, 2425, 2426, 2427, 2428, 2429, 2430, 2431, 2432, 2433, 2453, 2458, 2460, 2481, 2482, 2483, 2484, 2485, 2486, 2487, 2490, 2495, 2500
* (Chem): 2578, 2591, 2604
* (Immun): 2744, 2756, 2759, 2761, 2808
* (Path): 2847, 2875, 2884
* (Epid-Biom): 2924, 2944

2742 EMBRYONIC SURFACE ANTIGENS: A ''QUASI-ENDODERMAL'' TERATOMA ANTIGEN. (Eng.) Artzt, K. (Dept. Anatomy, Cornell Univ. Medical Coll., New York, NY 10021); Hamburger, L.; Jakob, H.; Jacob, F. *Dev Biol* 51(1): 152-157; 1976.

Antigens have been defined that appear to be stage-specific; i.e., their expression is limited to, and defines, cells in a particular early stage of differentiation. In parallel studies, hyperimmune antisera against a teratoma-derived endodermal carcinoma of 129/Sv SlJ C P mice were used in cytotoxicity tests and absorption analyses to define a surface antigen. This antigen was found on all tumor cells that were of endodermal origin as well as on embryonic liver, and was therefore designated ''Endo.'' Further testing, however, revealed that Endo was represented on several inappropriate cell types, such as young embryos before the appearance of endoderm, sperm, and a small proportion of nonendodermal tumors. Endo must therefore be considered a ''quasi-endodermal'' antigen. Only when more is known about the distribution of fetal substances such as Endo in normal embryonic tissues and in tumors of diverse origin will questions concerning the mechanism allowing their expression in malignant cells by appropriate. (19 references)

2743 EXPRESSION OF A CELL SURFACE ANTIGEN COMMON TO PRIMITIVE MOUSE TERATOCARCINOMA CELLS AND CLEAVAGE EMBRYOS DURING EMBRYOGENESIS AND SPERMATOGENESIS. (Eng.) Babinet, C.; Condamine, H.; Fellous, M.; Gachelin, G.; Kemler, R.; Jacob, F. In: *Teratomas and Differentiation*. Edited by M. I. Sherman and D. Solter. (New York: Academic Press, Inc.): pp. 101-107; 1975.

The expression of the cell surface F9 antigen, common to all primitive teratocarcinoma cells but not differentiated cells derived from teratomas, has been studied during normal embryogenesis and spermatogenesis with use of antisera produced by hyperimmunization of strain 129 male mice with irradiated syngeneic F9-41 teratocarcinoma cells. Unfertilized strain 129 mouse eggs had no detectable F9 antigen at the surface, but after fertilization with spermatozoa that bring traces of F9 antigen, the antigen was detected with the first cell division. The antigen was then expressed on all cells, even after differentiation into trophoblast and inner cell mass. Soon after implantation, the antigen disappeared from the surface of most cells of the embryo proper. On male germ cells, the F9 antigen was expressed not only on spermatozoa, but on all diploid precursors and gonocytes. This finding suggests that the F9 antigen is continuously expressed on germ cells, and thus on primordial gonocytes, at least in the male; the female germ line was not investigated. The expression of the F9 embryonic antigen and the absence of adult H-2 antigens in germinal cells may provide a way for the embryo to distinguish such cells from other differentiating tissues. (7 references)

2744 USE OF THE BLOCKING PROCEDURE IN MIXED HEMADSORPTION TESTS FOR THE ANALYSIS OF SURFACE ANTIGENS IN LIVER TISSUE CULTURE CELLS. (Eng.) Matveeva, V. A. (Inst. Experimental and Clinical Oncology, Acad. Medical Sciences, Moscow, USSR); Kluchareva, T. A.; Diechman, G. I. *J Immunol Methods* 9(3/4): 345-353; 1976.

Data concerning the possible use of blocking procedures in mixed hemadsorption tests (MHT) for detection of various species-specific and tumor-specific antigens in normal tissue culture, in live tumor cells, and in interspecies somatic hybrid cells are presented. Species specificity of the reaction was demonstrated with rabbit and guinea pig antisera against hamster, monkey, and mouse tissues. Absorption of species-specific antibodies from immune sera by corresponding cells showed a 4- to 8-fold decrease in titer in MHT after two cycles of absorption. The cell hybrid strain GMK-211 (a hybridization of hamster cells of SV40-induced tumor and the normal green monkey kidney cells) was studied in MHT with use of different antisera and blocking procedures. Species-specific antigens of green monkey and hamster origin were found on GMK-211 cell surfaces, but this was an impediment for detection of tumor-specific surface antigen using anti-GMK-211 serum. In blocking experiments, species-specific antigens were blocked by including pretreatment with appropriate antisera. To detect specific tumor antigens in SV40-transformed mouse embryo M-22 cells, the same immune serum could be used without a blocking procedure. A surface antigen that could not be blocked with the mixture of antihamster and antimonkey sera was discovered. The data demonstrate the possible use of unabsorbed heterologous sera to detect species-specific and tumor-specific surface antigens.

2745 PRESENCE OF BLOOD GROUP H ANTIGEN ON A CARCINOEMBRYONIC ANTIGEN, AND ITS ENZYMATIC MODIFICATION INTO BLOOD GROUP A AND B SPECIFICITIES. (Eng.) Bali, J. P. (Laboratoire de Biochimie II, Faculte de Pharmacie de Montpellier, France); Magous, R.; Lecou, C.; Mousseron-Canet, M. *Cancer Res* 36(7): 2124-2129; 1976.

A carcinoembryonic antigen (CEA-M) was purified from a hepatic metastasis obtained from a blood group O patient with cancer of the rectum. With the use of ^{125}I-labeled CEA, blood group antisera and the addition of human saliva extract (PES) to prevent the nonspecific adsorption of CEA on the immunoglobulin precipitate, H specificity was found on the CEA-M. Because the addition of anti-H to anti-CEA did not modify the extent of binding of labeled CEA-M to its antibodies (86%), the H and CEA determinants were carried by the same molecule. The affinity chromatography of CEA-M on an immunosorbent anti-H-Sepharose demonstrated that a proportion of CEA-M molecules might bear both H and CEA antigenic determinants. In addition, glycosyltransferases were used to modify the blood group H specificity into blood group A or B specificities. (21 references)

2746 TISSUE ANTIGENS AND CYTOTOXIC ANTIBODIES IN POLYCYTHAEMIA RUBRA VERA. (Eng.) Nagy, G. (Central Hosp. Robert Karoly krt. 44, 1134 Budapest, Hungary); Stenszky, V.; Timar, I.; Murvay, K. *Haematologia* 9(3/4): 279-282; 1975.

The incidence of ABO, Rh(D), and 22 tissue antigens and the development of antibodies against the three cell lines of hemopoiesis (erythropoiesis, myelopoiesis, and thrombocytopoiesis) were investigated in 46 polycythemia rubra vera (PRV) patients. There was no difference in ABO and Rh(D) levels compared to several thousand healthy controls. The tissue antigens, when compared to 490 controls, differed significantly only in an increase in HL-A5 and HL-A7 levels and a decrease in HL-A13 level. No erythrocytic, myelocytic, or thrombocytic antibodies were found. The lack of myelocytic antibodies did not support the hypothesis that moderate lymphopenia associated with PRV is due to lymphocytotoxic antibodies.

2747 ENDODERMALLY-DERIVED AND NEURAL CREST-DERIVED DIFFERENTIATION ANTIGENS EXPRESSED BY A HUMAN LUNG TUMOR. (Eng.) Bell, C. E., Jr. (Washington Univ. Sch. Medicine, St. Louis, MO 63110); Seetharam, S.; McDaniel, R. C. *J Immunol* 116(5): 1236-1243; 1976.

The plasma membrane antigens of an undifferentiated small cell (oat cell) carcinoma of the lung was studied by the indirect immunofluorescence method on frozen section substrates with a rabbit antiserum prepared to the tumor plasma membrane fraction. After absorption of the antiserum, at least two differentiation antigens were detected on the tumor cells but not on the normal lung surface or glandular epithelial cells. The first was characteristic of certain normal, endodermally derived epithelial cells of the digestive system, including those of the colonic mucosa, hepatic ducts, pancreatic ducts and acini, and islets of Langerhans. The second was characteristic of certain normal, neural crest-derived cells in the peripheral nervous system, including cells in the peripheral nerve, dorsal root ganglion, and anterior roots of the spinal cord; parasympathetic ganglion cells in the colon; and small nerves and nerve processes in the lung, colon, and skin. The presence of these differentiation antigens on the tumor cells was concluded to result from the expression of gene products repressed in the normal cell of origin of the tumor.

2748 DECREASED T-LYMPHOCYTES IN PATIENTS WITH MAMMARY CANCER. (Eng.) Keller, S. E. (Lenox Hill Hosp., 100 East 77th St., New York, NY 10021); Ioachim, H. L.; Pearse, T.; Siletti, D. M. *Am J Clin Pathol* 65(4): 445-449; 1976.

The levels of thymus-derived lymphocytes (T-cells) in samples of peripheral blood from 47 patients with carcinoma of the breast, 36 patients with benign mammary lesions, and 33 matched normal controls were determined. The results were analyzed using three statistical tests. The mean percentages of T-lymphocytes differed significantly among the three groups of patients ($p < 0.001$). The mean percentages of rosette-forming lymphocytes were 72.8 for the normal group, 60.6 for patients with benign mammary lesions, and 45.1 for the patients with carcinoma. The finding of lower levels of T-lymphocytes in the peripheral blood of individuals with mammary cancer is of particular significance since all the patients in this study were apparently in general good health and undergoing no treatment.

2749 DIFFERENTIATION AND MIGRATION OF T LYMPHOCYTES. (Eng.) Weissman, I. L. (Stanford Univ. Sch. Medicine, Stanford, CA); Masuda, T.; Olive, C.; Friedberg, S. H. *Isr J Med Sci* 11(12): 1267-1277; 1975.

The hypothesis that thymus cell migrants derive solely from the thymic medullary pool was tested by the characterization of Fischer 344 rat and C57BL/6J and BALB/c mouse thymus cell migrants in terms of their surface antigenic phenotypes, steroid sensitivity, and patterns of lymphoid tissue immigration. Thymocytes were selectively labeled in situ with a pulse incorporation of a radioactive nucleoside, and the animal was maintained under conditions of cold nucleoside "chase" until various tissues were removed for autoradiographic analysis. The proportion of medullary and cortisone-resistant (CR) thymocytes derived by recent cell division was higher in neonatal than in adult mice, these cells being replenished by cellular proliferation at both ages more slowly than cortical thymocytes. Fluorescent staining experiments with anti-Thy 1.2 and rabbit anti-mouse immunoglobulin demonstrated that thymus cell migrants possessed surface antigens characteristic of peripheral T cells. In other experiments, a subpopulation of mouse thymus cell migrants responded to trinitrophenol-keyhole limpet hemocyanin, allogeneic cells, and SRBC by transformation to large pyroninophilic cells; such a response was not seen in lymph nodes draining the site where *Salmonella adelaide* flagella (or saline) was injected. There was no evidence that thymus cell migration occurs in series through the spleen; rather, parallel migration streams were strongly implied. Under conditions of hydrocortisone (HC) treatment, residual mouse CR thymocytes did not contribute significantly to the emigrant pool. Furthermore, over 90% of recent thymus cell migrants were shown to be cortisone-sensitive cells in the periphery. It is concluded that CR thymocytes do not contribute disproportionately to the thymus cell migrant pool, that CR thymocytes cannot migrate in the presence of HC, or that, if they do, their peripheral descendants must rapidly regain cortisone sensitivity.

2750 T AND B LYMPHOCYTES AND REED-STERNBERG CELLS IN HODGKIN'S DISEASE LYMPH NODES AND SPLEENS. (Eng.) Payne, S. V. (Southampton Univ. Hosp., Tremona Road, Southampton SO9 4XY England); Jones, D. B.; Haegert, D. G.; Smith, J. L.; Wright, D. H. *Clin Exp Immunol* 24(2): 280-286; 1976.

Lymphoid cells from 24 untreated Hodgkin's disease biopsies were examined for spontaneous SRBC and sensitized ox erythrocyte rosette formation to identify T cells and cells with Fc and C3 receptors and surface immunoglobulin. Mean T lymphocyte values were elevated in both involved lymph nodes and uninvolved spleens from Hodgkin's patients, as compared to normal tissue. Lymphocytes bearing C3 receptors were correspondingly reduced in the Hodgkin tissues. Involved spleen T cell values fell within the normal range. In normal tissues the sum of lymphocytes with surface immunoglobulin and SRBC receptors fell in the range 89%-108%. This sum was outside the normal range (121%-142%) in six biopsies of Hodgkins tissues, which is compatible with surface immunoglobulin-coated T cells. Surface marker characteristics and intracellular immunoglobulin studies of small lymphocytes, lymphoblasts, and Hodgkin's cells suggest that the neoplastic cells were of B lymphocyte origin.

2751 C1 AND HUMAN PLATELETS. DECREASE IN CHRONIC LYMPHOCYTIC LEUKEMIA. EFFECT OF LYMPHOCYTES. (Eng.) Wautier, J.-L. (Hopital Lariboisiere, 2, rue Ambroise-Pare, 75475 Paris Cedex 10, France); Tobelem, G. M.; Peltier, A. P.; Caen, J. P. *Pathol Biol (Paris)* 23(Suppl): 33-36; 1975.

Studies on 11 patients with chronic lymphocytic leukemia and normal volunteers indicated that the platelet C1 hemolytic activity level was very low and that platelet aggregations induced by aggregated IgG or anti-C1q antisera were significantly reduced in the leukemia patients. The low C1 hemolytic activity may have been due to the absence of C1, its presence without hemolytic activity, or, it may have been present but inhibited. The reduced platelet aggregations did not seem to be due to a platelet abnormality, since platelet aggregation induced by ADP or collagen has been found to be normal. The results suggest a role of platelet C1 in the interaction of platelet/aggregated IgG. Furthermore, a lymphocytic origin of a factor reacting with C1 and associated with platelets could explain the impairment of platelet aggregation.

2752 SENSITIVITY OF TUMOUR AND NORMAL CELLS TO THE EFFECT OF IMMUNE LYMPHOCYTES IN VITRO AND SOME PROPERTIES OF TARGET CELLS DETERMINING THIS SENSITIVITY. (Eng.) Lavrovsky, V. A. (Inst. Cytology and Genetics, Siberian Branch Acad. Sci. USSR, Novosibirsk, USSR); Razvorotnev, V. A. *Folia Biol (Praha)* 22(2): 108-119; 1976.

Cells from various spontaneous mammary tumors of C3H/He mice differ in their sensitivity to immune allogeneic lymphocytes in vitro. Two cell lines with this difference were compared. Of the two, line MT3 was considerably more immunoresistant than line MT5, as demonstrated by microcytotoxicity tests in vitro. Furthermore, only the MT3 cell line was capable of growing in syngeneic hosts. During passages in vitro, MT5 cells became as immunoresistant as MT3 cells; concomitantly, MT5 cells started to take in syngeneic hosts. Cell immunosensitivity to immune allogeneic lymphocytes in vitro correlates with the cell property that determines the penetrance rate of intact allogeneic and syngeneic lymphocytes into these cells (emperipolesis). Nontumorous cells from different C3H/He mouse tissues were more immunoresistant than tumor cells from the two cell lines cultured. The studies demonstrated that various target cells differ significantly in the occurrence rate of specific interactions, and support the assumption that target cell immunosensitivity is determined not only by the quantity of antigen. (37 references)

2753 SERUM PROTEINS IN PROSTATIC CANCER: III. RELATIONSHIP OF LEVELS OF IMMUNOGLOBULINS AND COMPLEMENT TO CLINICAL STAGE OF DISEASE. (Eng.) Ablin, R. J. (Cook County Hosp., Chicago, IL 60612). *Urology* 7(1): 39-47; 1976.

The three major serum immunoglobulins (IgG, IgA, and IgM) and the third component of complement (C'3) were determined in the serum of 23 patients with prostatic carcinoma (5 Stage I, 7 Stage II, and 11 Stage III) and compared with values in normal males. Three patients had significantly decreased levels of IgG, 20 were normal, and none was elevated. Five had an elevated IgA level, 18 were normal, and one was depressed. Twenty-one patients had IgM below the normal mean, but this was not considered a significant decrease. Six patients had increased C'3, one had a decreased level, and the rest were in the normal range. The mean levels of Ig did not differ significantly among the various stages, but there was a significant difference between the mean level of each serum Ig of the patients and the controls ($p < 0.05$), indicating a decrease of IgG and IgM and an increase of IgA. The mean C'3 for all stages was significantly higher ($p < 0.05$) than for the healthy adults. Although there were no statistically significant differences according to stage, there was a trend upward from Stage I to Stage III. The absence of a correlation between the stage of disease and level of Ig is in keeping with observations of cell-mediated effectors of immunologic responsiveness in prostatic cancer patients. The association of prostatic cancer with a deficiency of B-cell function and of C'3 was noted.

2754 SUBFRAGMENTATION OF THE FC FRAGMENT OF HUMAN IGG1 MYELOMA PROTEIN BY THERMOLYSIN. (Eng.) Hunneyball, I. M. (Dept. Experimental Pathology, Univ. Birmingham, Birmingham B15 2TJ, England); Stanworth, D. R. *Immunology* 30: 579-586; 1976.

To determine which region of the human IgG molecule contains the antigenic determinant against which ''general'' (nonantiallotypic) rheumatoid factors are directed, the human myeloma protein IgG1 Fc fragment was digested at neutral pH by the enzyme thermolysin, from *Bacillus thermoproteolyticus* Rokko. Two large subfragments were produced: one, the tlmFc fragment, comprising the major part of the Fc fragment but devoid of the hinge region, appeared to consist of residues 235-446 of the γ chain; the second, the tlFc' fragment, comprised the Cγ3 domain. The former fragment retained the capacity to react with general rheumatoid factors, whereas the latter did not, indicating that the binding site for these factors on the Fc fragment of human IgG1 does not involve the hinge region of the molecule. (26 references)

2755 N-TERMINAL AMINO ACID SEQUENCE OF A HUMAN DELTA-CHAIN MYELOMA PROTEIN. (Eng.) Milstein, C. P. (A.R.C. Inst. Animal Physiology, Babraham, Cambridge CB2 4AT, England); Northrop, F. *Eur J Immunol* 6(3): 222-224; 1976.

The N-terminal sequence of a δ chain from immunoglobulin D (from human myeloma serum) was studied to determine whether its homology to other V region sequences was consistent with the idea that these chains share the same pool of V region genes as the rest of the immunoglobulin classes. The sequence (26 residues) was homologous with the prototype sequence of the VHIII subgroup, indicating that the δ chain is sharing the same pool of V genes.

2756 THE INFLUENCE OF THE THYMUS AND BURSA OF FABRICIUS IN CHICKENS WITH MAREK'S DISEASE (MD) INFECTIONS.

(Eng.) Shieh, H. K. (Paige Lab., Univ. Massachusetts, Amherst, MA 01002); Sevoian, M. *Bibl Haematol (Basel)* (43): 396-399; 1976.

The role of cellular and humoral immune systems in chickens in resisting Marek's disease (MD) was investigated with use of JM-P (JM-virus susceptible) and JM-N (JM-virus resistant) lines of Cornell White Leghorn chickens. Newborn chicks were thymectomized immediately after hatching and were injected ip immediately and on day 3 with anti-T serum. Some animals received thymus transplants from the opposite line of chicks. Ten days after surgery, JM virus (500 PFU/bird) was injected ip, and 4 and 6 wk later virus was recovered from the kidneys and quantitated. Both JM-P and JM-N thymectomized birds had higher virus levels ($p < 0.1$ and $p < 0.002$, respectively) at 4 wk than did sham-operated controls, and these differences were even more significant at 6 wk. JM-P chickens, which had been thymectomized at hatching and transplanted with JM-N thymus, had markedly lower virus levels than either thymectomized chicks ($p < 0.01$ at 4 wk, $p < 0.001$ at 6 wk) or sham controls ($p < 0.05$ at 4 wk and $p < 0.001$ at 6 wk). Resistant JM-N chickens receiving a JM-P thymic graft had about the same virus concentration as did thymectomized JM-N birds. Bursectomy within 24 hr after hatching or bursectomy followed by bursal graft from JM-P chickens did not influence MD virus replication in resistant JM-N birds. Virus levels in bursectomized or bursa-grafted JM-P birds were lower ($p < 0.4$ and $p < 0.2$) than those in the sham controls. The results suggest that the cellular immune system is involved in the protection against MD infection. (21 references)

2757 BIOASSAY FOR QUANTITATING CIRCULATING TUMOR CELLS IN A SYNGENEIC MOUSE TUMOR SYSTEM.

(Eng.) Lee, V. W. (Cleveland Clinic Foundation, Cleveland OH 44106); Chiang, T.; Deodhar, S. D. *Cancer Res* 36(6): 2053-2058; 1976.

A bioassay was developed to quantitate tumor cells in blood specimens in a syngeneic mouse tumor system (Sarcoma 1 in A/J mice). Blood containing tumor cells was injected im into each thigh of normal recipient mice and, 14 days later, the sites of injection were examined for evidence of tumor growth. A tumor index was calculated based on the number of tumor takes and the size of the tumors in each specimen. The number of tumor cells was determined by comparison with tumor indices from standard specimens with a known number of tumor cells. This bioassay was used to quantitate tumor cells in the venous blood of tumor-bearing mice under various treatments and manipulations. The incidence of regional node metastasis was also obtained by direct histological examination. Surgical removal of a well-established primary tumor enhanced the dissemination of the tumor, as evidenced by an increased incidence in regional node metastasis and an increase in the number of tumor cells reaching the venous circulation. Similar results were obtained when the tumor-bearing feet were ligated to produce ischemia of the primary tumor. Repeated physical trauma to the primary tumor resulted in increased dissemination of tumor cells into the venous circulation, but it did not increase the incidence of regional node metastasis. Immunosuppression of the tumor-bearing animals increased the dissemination of tumor cells, whereas immunostimulation decreased the dissemination.

2758 IMMUNODEFICIENCY IN HODGKIN DISEASE.

(Eng.) Eltringham, J. R. (Univ. Utah Medical Center, Salt Lake City, UT); Kaplan, H. S. *Birth Defects* 11(1): 278-288; 1975.

A study was made of the delayed hypersensitivity responses (DHR) of 218 untreated patients with Hodgkin's disease after sensitization with 2,4-dinitrochlorobenzene (DNCB) and a battery of intradermal antigens (purified protein derivative of tuberculin, blastomycin, mumps skin test antigen, *Candida albicans* extract, histoplasmin, and coccidiiodin). Also, a subgroup of patients who were not tested with blastomycin underwent id testing with streptokinase-streptodornase in addition to the other antigens. The use of suitable concentrations of antigens demonstrated immune deficiency in patients of all stages. For example, 83% of normal control subjects demonstrated positive DHR when challenged with a small dose of DNCB 5 days after sensitization by topical application of 0.1 ml of a 5% DNCB solution in acetone. Only 25% of Hodgkin's disease patients with Stages IA, IIA, or IIEA; 20% with Stages IIB or IIEB; 33% with Stages IIIA, IIISA, or IIIESA; 25% with Stages IIISB or IIIESB; or 25% with Stages IVA or IVB had positive reactions. Higher response rates were observed when high sensitizing concentrations of DNCB or streptokinase-streptodornase were employed. It is concluded that Hodgkin's disease patients at all stages have a depressed DHR: this impairment in cell-mediated immunity is a quantitative rather than an all-or-none phenomenon.

2759 PECULIARITY OF HUMORAL IMMUNITY IN BALB/C MICE WITH RAUSCHER LEUKEMIA.

(Rus.) Sokolov, P. P. (Moscow P. A. Gertsen Res. Inst. Oncology, Moscow, USSR); Bergol'ts, V. M. *Biull Eksp Biol Med* 81(3): 356-358; 1976.

BALB mice were challenged with plasma from BALB mice with developed Rauscher leukosis (0.1 ml, ip). In the second test group, 3 days prior to the Rauscher leukosis virus injection, the mice were stimulated with complete Freund's adjuvant (0.1 ml, ip). Surface immunoglobulins were detected by the immunofluorescent technique, and the antibodies against the surface antigens were revealed by the methods of nondirect immunofluorescence and cytotoxicity. It was shown that BALB/c mice highly sensitive to the Rauscher leukosis virus do not produce specific antibodies against Rauscher leukosis antigens. An increase in the number of spleen cells (B lymphocytes) carrying immunoglobulins on their surface was proved to be related to the primary immunological response. The results of the experiment confirmed the hypothesis that the observed surface immunoglobulins were not an actual product of the cell (cytophilic antibodies), but were simply extracellular immunoglobulins that attach to the lymphocyte. The spleens of mice stimulated with complete Freund's adjuvant exhibited both normal cells, which produced IgM antibodies, and leukosis cells, which adsorbed on their surface the blocking IgG antibodies. (10 references)

2760 ENHANCEMENT OF TUMOR GROWTH FOLLOWING IMMUNIZATION WITH *BACILLUS CALMETTE-GUERIN* CELL WALLS.

(Eng.) Wepsic, H. T. (Veterans Admin. Hosp., 3350 La Jolla Village Drive, San Diego, CA 92161); Harris, S.; Sander, J.; Alaimo, J.; Morris, H. *Cancer Res* 36(6): 1950-1953; 1976.

To determine whether an admixture of *Bacillus Calmette-Guerin* cell walls (BCGcw) and "soluble tumor antigen" could be an effective immunogen for the induction of tumor resistance, the effect of preimmunization with BCGcw on tumor growth was studied with transplantable Morris Hepatoma 3924a in inbred ACI rats 13 wk old. There was an increase in tumor incidence, in the im tumor growth rate, and in the average tumor mass in the animals. The BCGcw-induced enhancement in tumor growth depended on the amount of tumor present at the time of immunization. No enhancement occurred in animals immunized with BCGcw 12 days after the inoculation of tumor cells. The proposed mechanisms of tumor enhancement following immunostimulation are: antigenic competition may exist between BCG and the tumor; the induction of serum-blocking factors may occur in BCG-immunized animals; or immunostimulation by BCG and other related agents may alter the morphological structure and functional activity of lymphoid organs markedly. This immunostimulation may be a form of functional immunosuppression, the end result being an increase in the growth rate of any residual tumor and the subsequent clinical deterioration of the patient.

2761 EPSTEIN-BARR VIRUS (EBV) SPECIFIC T AND K CELL CYTOTOXICITY IN VITRO.

(Eng.) Jondal, M. (Dept. Tumor Biology, Karolinska Institutet, S-104 01, Stockholm, Sweden); Svedmyr, E.; Klein, E.; Klein, G. *Bibl Haematol (Basel)* (43): 265-269; 1976.

Studies directed toward demonstrating a cellular immune response to Epstein-Barr virus (EBV) as T cell immunity are reviewed. EBV-specific T cell cytotoxicity has been demonstrated in the blood of acute infectious mononucleosis (IM) patients, and preliminary experiments indicate the presence of this activity in the tumors but not in the blood of patients with Burkitt's lymphoma (BL) and nasopharyngeal carcinoma (NPC). Such activity is absent from the blood of both seropositive and seronegative normal individuals. EBV-specific K cell cytotoxicity is not present in the serum of acute IM patients or seronegative normal subjects, but it is present in the serum of patients with BL and NPC and seropositive normal individuals. Nonspecific cytotoxicity is present in the blood of all subjects tested. The presence of EBV DNA in tumor cells derived from BL and NPC patients has been clearly demonstrated by hybridization studies. These tumor cells also express the EBV-induced nuclear EBNA antigen. EBNA-positive cells are present in the acute phase of IM and disappear rapidly after that. EBNA-positive cells are present in only small amounts (1%-2%) and are limited to the B cell population. The findings support the immune surveillance concept. (28 references)

2762 GROWTH CHARACTERISTICS OF A HUMAN BLADDER TUMOUR SUBCUTANEOUSLY IMPLANTED IN IMMUNE DEFICIENT MICE.

(Eng.) Franks, C. R. (Guy's Hosp., London, SEI 9RT, England); Turner, D. R.; Bishop, D.; Perkins, F. T. *Clin Oncol* 2(1): 25-32; 1976.

A human transitional cell bladder carcinoma, on its second passage, was implanted sc in immunodeficient female CBA mice. Growth was assessed by histological analysis, following needle biopsy performed at 14-day intervals, and also by measuring the transverse and vertical diameters of the palpable tumor. Between days 20 and 25 after implantation, the tumor appeared to undergo a process of selection, which subsequently determined whether there was an increase in size or regression.

2763 TRANSPLANTATION OF TUMOURS.

(Eng.) Metcalf, D. (Hall Inst., Melbourne, Australia). *Int Union Cancer Tech Rep Ser* 20: 40-43; 1975.

General aspects of tumor transplantation are discussed. Transplantation may be used to verify the neoplastic nature of an abnormal population, or to determine: the genotype of tumor cells arising in a chimeric animal; the histological type of the original tumor; the dependency of transplanted tumor cells on endocrine or regulator imbalances; the antigenicity of tumor cells; and immunological, hematological, or metabolic changes occurring in tumor-bearing animals. Transplanted tumors may be used as initial screening tests for anti-tumor agents. Tumor transplantation should always be performed with tissue from animals killed immediately before removal of the tissue. Tumors vary with respect to the number of cells required for successful transplantation, depending on the number of previous tumor transplantation passages and the tumor type. Common sites of injection are the subcutaneous tissues over the flanks or behind the neck, the thigh muscles, or intraperitoneal sites. No transplantation site is known to be preferable to others in terms of permitting growth of transplanted tumor cells. Ideally, the tumor cells should be placed in a location where progressive tumor growth is detectable by palpation or caliper measurement and where the growth of transplanted tumor cells cannot be confused with a spontaneously developing tumor. Most tumors transplanted (sc or im) form local tumors which may or may not disseminate. Leukemic cells or reticulum cell sarcoma cells injected ip commonly produce generalized involvement of lymphoid organs rather than local intraperitoneal tumors. Most transplanted tumors form obvious tumors in the recipients within 3-4 wk, but the growth of tumors in the first transplant generation may be very slow. In the test of an unknown or potential tumor, mice should not be discarded as negative before at least 6 mo after injection of tumor cells. An animal bearing a transplanted tumor has not ''developed'' cancer; this should be remembered in interpretation of results.

2764 ARE DEAD CELLS RESPONSIBLE FOR ALLOGENEIC GROWTH OF ORGAN CULTURES AND CELL SUSPENSIONS OF BALB/C LEYDIG CELL TUMORS? (Eng.) Jacobs, B. B. (American Medical Center at Denver, Spivak, CO 80214). *Transplantation* 21(5): 433-436; 1976.

The proposal that allogeneic growth of the BALB/c Leydig cell tumor C4092 following cell dispersal or explantation may be due to immunological enhancement induced by solubilized antigens released from the high proportion of dead cells present was tested. The addition of heat-killed, freeze-thaw-killed, or irradiated cells by the sc, iv, or ip route did not significantly increase the growth of solid fragments of the strain-specific BALB/c C4092 tumor in DBA/1 mice. Evidence of an alternative explanation, that allogeneic acceptance of such tumors following explantation of cell suspension is due to phenotypic reduction of immunogenicity possibly associated with a configurational change in the cell membrane, is reviewed.

2765 IMMUNOLOGICAL ENHANCEMENT OF A MURINE TUMOR ALLOGRAFT BY PASSIVE ALLOANTIBODY IgG AND $F(AB')_2$. (Eng.) Kaliss, N. (Jackson Lab., Bar Harbor, ME 04609); Sinclair, N. R.; Cantrell, J. L. *Eur J Immunol* 6(1): 38-42; 1976.

Studies were conducted on an enhancement system in inbred male mice of the C57BL/Ks (H-2d) strain (BKs) to determine if the Fc portion of an alloantibody IgG, directed against a tumor allograft, is an essential participant in suppressing the cellular anti-tumor rejection response. $F(ab')_2$ was isolated from seven BKs anti-SaI sera. Five 30-wk-old mice in each group were given an ip injection of either 3.4 mg IgG or 0.2 ml of whole BKs anti-SaI serum. Another three-mouse group received 3.6 mg, and three mice received 0.2 mg of $F(ab)_2$. Tumor cells (A(H-2a) sarcoma I: (SaI); 3 x 10^6 cells, im) were injected 30 min later into the calf muscle. In the first experiment, tumor growth rates were highest in the groups given IgG or whole serum. In the group given 3.6 mg $F(ab'0)_2$ there was an increase in calf size by day 7, followed by a decrease by day 15 and a subsequent recurrence of growth by day 30. This may represent autoenhancement by indigenous anti-tumor antibody developed in response to the tumor inoculum. In another experiment, groups of mice (5/group) were treated after tumor inoculation. Treatment was: undigested IgG, $F(ab')_2$ + IgG, $F(ab')_2$, IgG, and ''control pepsin digest'' (serum carried through $F(ab')_2$ isolation without added pepsin). All treated groups had tumor enhancement compared to untreated controls. Multiple injections of $F(ab')_2$ after grafting were nearly as potent as IgG or whole serum, indicating that the absence of the Fc moiety of IgG did not impair its enhancing activity. In contrast to the results with tumor enhancement, $F(ab')_2$ was less depressive of the humoral response to allogeneic spleen cells than IgG or whole antibody.

2766 IMMUNOLOGICAL PROPERTIES OF A HOMOGENEOUS SJL/J RETICULUM CELL SARCOMA (Proceedings Abstract). (Eng.) Owens, M. H. (Dept. Microbiology and Immunology, UCLA Sch. Medicine, Los Angeles, CA 90024). *Proc Am Assoc Cancer Res* 17: 217; 1976.

2767 IMMUNOLOGICAL STUDIES IN REGIONAL LYMPH NODES IN HUMAN MALIGNANT MELANOMA (Proceedings Abstract). (Eng.) Lewis, M. G. (McGill Univ. Cancer Res. Unit, Montreal, Quebec, Canada); Rowden, G.; Shibata, H.; Jerry, L. M.; Shea, M.; Lyons, H.; Quirk, C. *Proc Am Assoc Cancer Res* 17: 141; 1976.

2768 IMMUNOLOGICAL RELATIONSHIP OF HUMAN BREAST CARCINOMA AND BENIGN BREAST DISEASE AS DETECTED BY LEUKOCYTE MIGRATION INHIBITION (Proceedings Abstract). (Eng.) Cannon, G. B. (Litton Bionetics, Inc., Kensington, MD 20795); McCoy, J. L.; Nyirjesy, I.; Alford, C.; Herberman, R. B. *Proc Am Assoc Cancer Res* 17: 136; 1976.

2769 IMMUNE STATUS OF MICE DURING CARCINOGENESIS BY ULTRAVIOLET RADIATION (Proceedings Abstract). (Eng.) Kripke, M. L. (NCI-Frederick Cancer Res. Center, Frederick, MD 21701); Lofgreen, J. S. *Proc Am Assoc Cancer Res* 17: 49; 1976.

2770 IN VITRO AUGMENTATION OF TUMOR IMMUNITY IN A MURINE METHYLCHOLANTHRENE SARCOMA SYSTEM (Proceedings Abstract). (Eng.) Yarlott, M. A. (Univ. Minnesota, Minneapolis, MN 55455); Kasperson, E. H.; McKhann, C. F. *Proc Am Assoc Cancer Res* 17: 46; 1976.

2771 HUMORAL IMMUNE FACTORS IN RESISTANCE TO SOLID TUMOR GROWTH (Proceedings Abstract). (Eng.) Vaage, J. (Dept. Cancer Therapy Development, Pondville Hosp., Walpole, MA 02081); Agarwal, S. *Proc Am Assoc Cancer Res* 17: 1; 1976.

2772 MECHANISMS OF CELL-MEDIATED IMMUNE STIMULATION AND INHIBITION (Proceedings Abstract). (Eng.) Norbury, K. C. (NCI-Frederick Cancer Res. Center, Frederick, MD 21701). *Proc Am Assoc Cancer Res* 17: 145; 1976.

2773 SELECTIVE CELL-MEDIATED CYTOTOXICITY (Proceedings Abstract). (Eng.) Flashman, R. (Univ. California, Los Angeles, CA 90024); Takasugi, J. *Proc Am Assoc Cancer Res* 17: 69; 1976.

2774 LACK OF CORRELATION OF ^{86}Rb RELEASE AND CELL DEATH IN THE ANTIBODY-COMPLEMENT MEDIATED KILLING OF A TUMOR CELL (Proceedings Abstract). (Eng.) Boyle, M. D. P. (Natl. Insts. Health, Bethesda, MD 20014). *Proc Am Assoc Cancer Res* 17: 162; 1976.

2775 LECTIN MEDIATED CELLULAR CYTOTOXICITY AGAINST HUMAN MELANOMA CELLS (Proceedings Abstract). (Eng.) Golub, S. H. (UCLA Div. Surgical Oncology, Los Angeles, CA 90024). *Proc Am Assoc Cancer Res* 17: 90; 1976.

2776 FURTHER CHARACTERIZATION OF IMMUNOSPECIFIC DNA-PROTEIN COMPLEXES (Proceedings Abstract). (Eng.) Chiu, J.-F. (Dept. Biochemistry, Vanderbilt Univ., Nashville, TN 37232); Fujitani, H.; Hnilica, L. S. *Proc Am Assoc Cancer Res* 17: 188; 1976.

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2801 A NEW ONCOFETAL ANTIGEN ASSOCIATED WITH GASTROINTESTINAL CANCER (Proceedings Abstract). (Eng.) Goldenberg, D. M. (Dept. Pathology, Univ. Kentucky Medical Center, Lexington, KY 40506); Pant, K. D.; Dahlman, H. *Proc Am Assoc Cancer Res* 17: 155; 1976.

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2817 T LYMPHOCYTES IN CHRONIC LYMPHATIC LEUKEMIA AND CHANGES IN THE GENETIC ACTIVITY. (Fre.) Conte, P. F. (Instituto de Semeiotica Medica, Universita di Torino, Turin, Italy); Masera, P.; Pileri, A. *C R Acad Sci (D) (Paris)* 281(17): 1289-1292; 1975.

2818 THE T LYMPHOCYTE SURFACE IN DEVELOPMENT: A STUDY OF THE ELECTROKINETIC, ANTIGENIC AND ULTRASTRUCTURAL PROPERTIES OF T LYMPHOCYTES IN MOUSE THYMUS AND LYMPH NODES. (Eng.) Zeiller, K. (Max Planck Inst. Biochemistry, Martinsried near Munich, W. Germany); Schindler, R. K.; Liebich, H. G. *Isr J Med Sci* 11(12): 42-66; 1975.

2819 GENERATION OF CYTOTOXIC LYMPHOCYTES IN MIXED LEUKOCYTE CULTURE BY RAT LYMPHOID CELLS. (Eng.) Bernstein, I. D. (Fred Hutchinson Cancer Res. Center, Seattle, WA 98104); Wright, P. W. *Transplantation* 21(2): 173-178; 1976.

2820 SEPARATION OF POPULATIONS OF SENSITIZED LYMPHOID CELLS INTO FRACTIONS INHIBITING AND FRACTIONS ENHANCING SYNGENEIC TUMOR GROWTH IN VIVO (Proceedings Abstract). (Eng.) Small, M. (Dept. Cell Biology, Weizmann Inst. Science, Rehovot, Israel); Trainin, N. *Proc Am Assoc Cancer Res* 17: 213; 1976.

2821 REVERSAL OF LYMPH NODE REFRACTORINESS IN MICE BEARING LARGE TUMORS (Proceedings Abstract). (Eng.) Burk, M. W. (Univ. Minnesota, Minneapolis, MN 55455); Yu, S. *Proc Am Assoc Cancer Res* 17: 152; 1976.

See also:

* (Rev): 2427, 2429, 2433, 2434, 2435, 2436, 2437, 2438, 2439, 2440, 2441, 2442, 2443, 2445, 2446, 2458, 2481, 2488, 2490, 2491, 2492, 2493, 2494, 2495, 2496, 2499
* (Chem): 2520, 2534, 2544, 2594, 2595
* (Viral): 2638, 2643, 2645, 2650, 2652, 2656, 2657, 2675, 2679, 2685, 2686, 2710, 2717, 2719, 2720, 2729, 2735
* (Path): 2825, 2827, 2831, 2834, 2844, 2848, 2875, 2879, 2880, 2882, 2902, 2919, 2920
* (Epid-Biom): 2950

2822 GIANT PLATELET GRANULES IN REFRACTORY ANEMIA (PRELEUKEMIA) AND MYELOMONOCYTIC LEUKEMIA: A CELL MARKER? (Eng.) Maldonado, J. E. (Mayo Clinic, 200 First St. SW, Rochester, MN 55901). *Blood Cells* 1(1): 129-135; 1975.

The ultrastructural study of the platelets of 16 patients with refractory anemia (''preleukemia'') or myelomonocytic leukemia revealed several abnormalities relating to changes in size and shape of the platelet as well as alterations in the subcellular organelles, including abnormalities in the number, size, contour, and internal structure of the platelet granules. Particularly striking were the giant granules (0.5 to 2.5 μm in widest diameter) that were apparently the result of the ''fusion'' of several single granules. These structures, which have not been found in other pathologic states, seem to be derived primarily from the α or specific granules and are visible by light microscopy. They may constitute a cell marker of the platelets in preleukemia and myelomonocytic leukemia. These granules probably participate less actively than the normal granules during platelet aggregation. Some remain intact in advanced stages of platelet aggregation and degranulation.

2823 CYTOGENETIC STUDIES IN PRELEUKEMIA: STUDIES BEFORE AND AFTER TRANSITION TO ACUTE LEUKEMIA IN 17 SUBJECTS. (Eng.) Pierre, R. V. (Mayo Clinic, 200 First St. SW, Rochester MN 55901). *Blood Cells* 1(1): 163-170; 1975.

In a prospective cytogenetic study of 205 patients with suspected preleukemia, 131 had normal bone-marrow chromosome values and 74 had abnormal ones; 32 patients of the 205 have developed overt acute leukemia to date. The incidence and type of chromosome abnormalities observed in these 32 transformed patients were similar to those observed in other patients with overt acute leukemia. Serial, direct, bone-marrow chromosome findings in 17 of the 32 patients during the preleukemic and leukemic phases of their illnesses revealed four cytogenetic patterns: normal initial findings that remained normal; normal initial findings and a clonal abnormality developing coincident with the transition to acute leukemia; abnormal initial findings that remained stable; and abnormal initial findings with a new abnormality developing at the time of transition to acute leukemia. The presence of normal, direct, bone-marrow chromosome findings does not eliminate the possibility that a disorder is truly a preleukemic disorder. Nor does the presence of a clonal chromosome abnormality constitute proof that a patient's disorder is preleukemic, since clonal abnormalities may be observed in chronic myeloproliferative disorders such as agnogenic myeloid metaplasia, polycythemia vera, and chronic granulocytic leukemia. Cytogenetic studies suggest that in some patients, the preleukemic phase is a distinct entity and not an early phase of acute leukemia. Patients with initial cytogenetic abnormalities have a poorer prognosis.

2824 GENETICS SYNDROMES AND PREDISPOSITION TO MALIGNANT NEOPLASMS. (Eng.) Swift, M. (Univ. North Carolina Sch. Medicine, Chapel Hill, NC). *Int J Dermatol* 14(10): 733-734; 1975.

In eight families of Fanconi anemia patients, a significant increase in deaths from cancer and leukemia was observed. In a study of 27 families of patients with ataxia-telangiectasia, there was a highly significant increase in deaths from malignant neoplasms in blood relatives dying below age 75 and an even greater increase below age 45. It was estimated that the risk of a person heterozygous for the disease dying of cancer before age 45 is five times greater than that for an individual in the general population. The risk of death from cancer between ages 45 and 75 is twofold over the general population. The associated malignancies are those of the lymphopoietic system and ovarian, gastric, biliary system, breast, and cervical carcinomas. Heterozygotes for the ataxia-telangiectasia gene comprise about 1% of the population, but there is no test to detect them. Members of such families should undergo regular checkups aimed at detecting neoplasms. Families of patients with other genetic syndromes predisposing to cancer, such as xeroderma pigmentosum, may also be at risk for developing cancer; physicians are advised to report relevant findings to active investigators in the field.

2825 ACUTE BLASTIC TRANSFORMATION IN PATIENTS WITH CHRONIC GRANULOCYTIC LEUKAEMIA AND MYELOFIBROSIS. (Eng.) Bernstein, R. (Dept. Haematology, Univ. Witwatersrand, Johannesburg, South Africa); Katz, J.; Bezwoda, W.; Lynch, S. R. *S Afr Med J* 50(15): 616-620; 1976.

A retrospective study of 21 patients with acute transformation of chronic granulocytic leukemia (CGL) and myelofibrosis was undertaken. The patients were divided into 3 groups according to whether transformation had occurred in pre-existing CGL (11 patients), myelofibrosis (6 patients), or in patients who presented with acute leukemia and significant marrow fibrosis--acute myelofibrosis (4 patients). To determine whether, during the acute blastic crisis, there is conversion to the hemopoietic stem cell, the morphology of the blast cell and the response to therapy were assessed in 17 of the 21 patients. Three patients had blast cells that morphologically resembled lymphoblasts; their mortality was similar to that in patients in whom the blast cell morphology was myeloblastic. The response to treatment in the acute phase was poor. However, some patients who received therapy with thioguanine, daunorubicin, cytosine arabinoside, methotrexate, prednisone, cyclophosphamide, and vincristine (TRAMPCO) apparently improved. In four patients, splenectomy was performed for advanced disease; acute blastic transformation supervened within 4 mo in three of these patients. In three of five patients with myelofibrosis, splenectomy was followed by an increase of primitive cells in the peripheral blood and rapid liver enlargement. Although they had improved symptomatically, the patients died within 10 mo of the splenectomy. The indications for and timing of splenectomy remain controversial.

2826 TWO COMPLEX TRANSLOCATIONS IN CHRONIC GRANULOCYTIC LEUKEMIA INVOLVING CHROMOSOMES 22, 9, AND A THIRD CHROMOSOME. (Eng.) Nowell,

P. C. (Univ. Pennsylvania Sch. Medicine, Philadelphia, PA); Jensen, J.; Gardner, F. *Humangenetik* 30(1): 13-21; 1975.

Among 13 Ph-positive cases of chronic granulocytic leukemia (CGL), banding studies revealed 2 with complex rearrangements involving translocation of the long arm of number 22 to another autosome and a segment of that chromosome translocated to the long arm of number 9. In a patient (black male) with both CGL and sickle cell anemia, the three-way rearrangment involved chromosomes 5, 9, and 22. He also had a second Philadelphia chromosome and two constitutional variants: pericentric inversion of the other number 9 chromosome and satellite polymorphism in the G group. The karyotype of the leukemia cells was interpreted as: 47, XY, inv(9) (p11q13), t(5;9;22) (q13;q34;q11) + del(22) (q11). In the second patient (white female), the complex translocation in the Ph-positive cells involved chromosomes 3, 9, and 22, resulting in a karyotype interpreted as: 46, XX, t(3;9;22) (p21;q34;q11). Eleven patients had the usual translocation 9qt;22q-. Several reports indicate that an abnormality of chromosome 9 is not essential for the development of Ph-positive CGL, but the very high frequency of its involvement (including these unusual translocations) suggests that some type of nonrandom somatic association may exist between 9q and 22q in interphase that makes simultaneous breakage likely.

2827 FAMILIAL CHRONIC LYMPHOCYTIC LEUKEMIA. IMMUNOLOGIC AND CELLULAR CHARACTERIZATION. (Eng.) Blattner, W. A. (Natl. Cancer Inst., Bethesda, MD); Strober, W.; Muchmore, A. V.; Blaese, R. M.; Broder, S.; Fraumeni, J. F., Jr.; *Ann Intern Med* 84(5): 554-557; 1976.

Four siblings with chronic lymphocytic leukemia were studied by use of laboratory probes to define cell-surface immunoglobulins and other immunologic variables. The disease occurred in four of five siblings whose father also was affected. Absolute peripheral levels were normal except for borderline low levels of IgM in three and IgA in one. All five sibs responded to at least one antigen in a battery of delayed hypersensitivity skin tests. In vitro proliferative responses of peripheral lymphocytes to nonspecific mitogens, specific antigens, and allogeneic lymphocytes in mixed WBC culture were abnormal in the four affected sibs. The degree of dysfunction paralleled the severity of the disease. All four also had marked impairment of cytotoxicity function. Staining of the peripheral blood lymphocytes with specific fluorescent anti-Ig reagents showed that over 50% of the lymphocytes bore δ and κ chains but not μ, γ, or α chains in three of the affected sibs. The fourth bore no detectable Ig, and the normal sibling had lymphocytes with normal proportions of all chain types. The results indicate that familial susceptibility to chronic lymphocytic leukemia is inherited through a specific class of cells, apparently those destined to express δ heavy and κ light chains in this family.

2828 G-G TRANSLOCATION IN ACUTE MYELOCYTIC LEUKEMIA WITH LOW NEUTROPHIL ALKALINE PHOSPHATASE ACTIVITY. (Eng.) Kamada, N. (Res. Inst. Nuclear Medicine and Biology, 1-2-3, Kasumi-cho, Hiroshima City, Japan, 734); Okada, K.; Oguma, N.; Tanaka, R.; Mikami, M.; Uchino, H. *Cancer* 37(5): 2380-2387; 1976.

Sequential cytogenetic analysis of bone marrow cells and neutrophil alkaline phosphatase (N-AL) activity before treatment, in remission, and in relapse was performed on 65 patients with adult myelocytic leukemia (AML); morphologic abnormalities of blood cells and survival time were also studied. A consistent chromosome abnormality of C-G translocation, t(8;21)(q22;q22), was found in 15 patients with low N-AP activity. Granulocytes of these patients also had specific morphologic abnormalities (decreased number of granules, eccentric localization of granules in neutrophils, Auer rods in mature neutrophils, peroxydase negative neutrophils, Pelgerlike anomaly, and relatively good maturation in the granulocyte series). The bone marrow showed a tendency to relatively good maturation of leukemic cells for the disease AML. The clinical courses of the patients were mild and median survival was longer than that of patients with normal or high N-AP activity. Three out of six male patients with this type of AML had a missing Y chromosome in addition to C-G translocation. The results suggest that specific cytogenetic abnormality of C-G translocation is significantly associated with AML. Because these abnormalities differ from those of blastic crisis of chronic myelocytic leukemia, it is suggested that chronic myelocytic leukemia patients form a subgroup of AML.

2829 VARIATIONS OF THE PROLIFERATIVE ACTIVITY OF LEUKEMIC MYELOBLASTS RELATED TO THE STAGE OF THE DISEASE. (Eng.) Stryckmans, P. (Institut Jules Bordet, 1, rue Heger-Bordet, 1000 Brussels, Belgium); Debusscher, L.; Peltzer, T.; Socquet, M. *Blood Cells* 1(1): 239-248; 1975.

To determine whether the low proliferative activity of bone marrow myeloblasts of acute myeloblastic leukemia (AML) is related to the number of myeloblasts through some regulatory mechanism, the myeloblasts of 26 AML patients in complete remission (CR) or in the active phase and those of 16 in relapse or in remission of chronic myeloid leukemia (CML), and 10 hematologically normal reference patients were studied by tritiated thymidine (^{3}HTdR) and autoradiography. In AML, the ^{3}HTdR labeling index of the myeloblasts returned to normal values and remained within that range during complete remission in six patients examined. In CML, the ^{3}HTdR labeling index of the myeloblasts but not of more mature myeloid cells was normal when the WBC was below 20,000/mm^3, but it decreased when the WBC increased above 20,000/mm^3. It is concluded that the low labeling indices of the myeloblasts during active AML and CML do not indicate the ''leukemic nature'' of these cells but rather reflect the activity of regulatory mechanisms probably operating on the normal myeloblast and persisting somehow during leukemia.

2830 UNREGULATED GROWTH OF MURINE LEUKEMIC CELLS AND SUPPRESSION OF NORMAL GRANULOCYTE GROWTH IN DIFFUSION CHAMBER CULTURES.

(Eng.) Miller, A. M. (Beth Israel Hosp., 330 Brookline Ave., Boston, MA 02215); Marmor, J. B.; Page, P. L.; Russell, J. L.; Robinson, S. H. *Blood* 47(5): 737-745; 1976.

The malignant growth of leukemic cells, specifically the patterns of proliferation of C1498 mouse acute leukemic cells, were studied in diffusion chamber cultures. Irrespective of initial cell input, all the malignant cells grew to the same maximal cell concentration. Leukemic cells proliferated equally well with or without the stimulus of prior host irradiation. When cells cultured for several days were diluted to the original input concentration and recultured, they rapidly proliferated back to maximal cell number. All of these findings are in sharp contrast to the behavior of granulocytes from normal mouse marrow grown in the same culture system. Coculture of equal numbers of normal marrow cells and leukemic cells resulted in virtually complete inhibition of normal granulocyte growth.

2831 MALIGNANT LYMPHOMA OF THE SMALL INTESTINE IN MULTIPLE FAMILY MEMBERS: ASSOCIATION WITH AN IMMUNOLOGIC DEFICIENCY.

(Eng.) Maurer, H. S. (Children's Memorial Hosp., 2300 Children's Plaza, Chicago, IL 60614); Gotoff, S. P.; Allen, L.; Bolan, J. *Cancer* 37(5): 2224-2231; 1976.

Four cases of familial malignant lymphoma (three of five brothers and their sister's son, all of whom developed the disease as children within 7 yr of each other) are described. In three of the affected individuals the primary tumor arose in the small intestine, while in one brother the site of origin was the retroperitoneum. Immunologic studies of the two surviving patients (oldest brother and nephew) and on all other living members of their families revealed low levels of immunoglobulins in the brother. During a recurrence of the tumor, the nephew showed temporary partial impairment of cellular immunity, measured by failure to react to delayed hypersensitivity skin tests; lymphocyte transformation studies, however, were normal. Nine months after treatment his response to skin tests was normal. No immunologic defect was detected in studies of the noninvolved family members. The histologic appearance of the tumors was similar in the tissue sections from three of the four patients; the tumor was classified as a malignant lymphoma, diffuse, mixed cell type (lymphocytic and histiocytic). This occurrence of malignant lymphomas in multiple male members of a family (with hypogammaglobulinemia in one of the patients) suggests that genetic and immunologic factors may play a role in its development.

2832 HEPATIC ANGIOSARCOMA IN WORKERS FOLLOWING CHRONIC EXPOSURE TO VINYL CHLORIDE. MORPHOLOGICAL DESCRIPTION OF THE LESIONS.

(Fre.) Delorme, F. (Centre hospitalier regional de la Mauricie, C.P. 1130, Shawinigan-Sud, Quebec, Canada); Makk, L. *Union Med Can* 104(12): 1836-1844; 1975.

The histological characteristics of liver angiosarcomas (2 metastases) in 18 men (36 to 58 yr; mean age 46.7) who had prolonged exposure to vinyl chloride (4 yr to 27 yr 10 mo; mean time 17 yr 4 mo) are presented. Various benign lesions were noted not only in the angiosarcoma cases but also in noncancerous cases who also had prolonged exposure. Many of the perisinusoidal cells of the angiosarcomas resembled, with their elongated nuclei, the small mesynchymatous cells of the benign tumors. The angiosarcomas had different forms: sinusoidal, sarcomatous, nodulopapillary, and cavernous. These cancers are generally preceded by the appearance of the benign lesions induced by the vinyl chloride. These lesions appear simultaneously in many places and apparently are multifocal. The numerous nodules are not intraperenchymal metastases from a primary tumor but are themselves primary tumors.

2833 RETICULUM CELL SARCOMA OF THE CECUM AND MACROGLOBULINEMIA: A CASE REPORT.

(Eng.) Levy, M. (Long Island Jewish-Hillside Medical Center, New Hyde Park, NY 11040); Stone, A. M.; Platt, N. *J Surg Oncol* 8(2): 149-153; 1976.

The case report of an 81-year-old man who developed a reticulum cell sarcoma of the cecum while in immunosuppressive therapy is presented. The man had been on immunosuppressive therapy for 13 yr for a primary diagnosis of macroglobulinemia. This unusual case suggests the possible relationship of the development of a malignancy in patients with an immunological disease further compounded by immunosuppressive therapy, and illustrates the importance of aggressive followup in this type of patient.

2834 KARYOTYPES OF VASOFORMATIVE SARCOMAS ARISING FROM BALB/3T3 CELLS ATTACHED TO POLYCARBONATE PLATES.

(Eng.) Yoshida, M. C. (Faculty Science, Hokkaido Univ., Sapporo, Japan); Sasaki, M.; Takeichi, N.; Boone, C. W. *Cancer Res* 36(7): 2235-2240; 1976.

The quinacrine banding technique was used to analyze the chromosomes of four vasoformative sarcoma in vivo tumor lines arising from the sc implantation of 3 x 10^4 BALB/3T3 cells attached to 1 x 5 x 10 mm polycarbonate platelets. These lines were shown to be quite similar in chromosome constitution to the parental 3T3 line. All tumors contained the same marker chromosomes present in the parent cells. The findings conclusively showed that the tumors had derived from BALB/3T3 cells and not from host cells or from the in vivo fusion of the inoculated 3T3 cells with host cells. Each tumor line had a unique karyotype that was much more homogeneous than that of the parental cells, strongly suggesting that each tumor represented a separate clone. An M1 marker chromosome was consistently present in each of the four tumor lines but was present in only about 50% of the parent BALB/3T3 cells; it therefore appeared to be a distinct and stable feature of the tumors. One tumor line acquired an isochromosome of the 1/1 centric fusion, which must have formed during in vivo progression; the line was positive only for oncornaviruses and reverse transcriptase. Since homology association is frequently seen in mouse cell chromosomes, it is not clear whether the formation of the 1/1 centric fusion is related to the presence of endogenous viruses. (22 references)

2835 KAPOSI'S DISEASE: ULTRASTRUCTURAL STUDY OF 4 CASES. (Fre.) Bonerandi, J.-J. (Hotel-Dieu, 6, place Daviel, F 13324 Marseille Cedex 1, France); Bourdet, D.; Privat, Y.; Temime, P. *Ann Dermatol Syphilgr (Paris)* 102(5-6): 499-506; 1975.

The ultrastructural study of four Kaposi's sarcomas showed that the tissue was essentially composed of immature vascular formations; these were vascular slits and not true vessels. These slits were nonuniformly bordered by either globular or fusiform endothelial cells. There were many inflammatory cells (lymphocytes, plasmocytes, histocytes, and macrophages), fibroblasts, and pericytes. The globular and fusiform endothelial cells and the macrophages all participated equally in the phagocytosis of the numerous effused erythrocytes. There was no evidence of cytoplasmic or intranuclear viral particles. The disease was probably the result of a reticuloendothelial proliferation with multiple morphological types and predominant vascular differentiation.

2836 GENETIC HETEROGENEITY AND FAMILIAL CARCINOMA OF THE BREAST. (Eng.) Lynch, H. T. (Creighton Univ. Sch. Medicine, Omaha, NB); Gurgis, H. A.; Brodkey, F.; Lynch, J.; Maloney, K.; Rankin, L.; Mulcahy, G. M. *Surg Gynecol Obstet* 142(5): 693-699; 1976.

The distribution of site-specific carcinoma of the breast and carcinoma of the breast in association with several other histologic varieties in 52 families prone to this disease is described. The mean number of persons per family was 36.8. Twenty-nine families showed predominantly site-specific carcinoma of the breast, and 23 families showed several different patterns of tumor associations. An analysis of person-years at risk and age-specific incidence of cancer of all anatomic sites in members of three of these families indicated a significant ($p < 0.001$) increase in cancer frequency when compared to the New York State Tumor Registry. Physicians caring for persons from families with a history of breast carcinoma should be alert to the increased risk for this cancer as well as that of other anatomic sites and should be aware of the possible earlier onset of cancer in these patients.

2837 ENDODERMAL SINUS TUMOUR OF THE OVARY: A COMPARATIVE LIGHT AND ELECTRON MICROSCOPIC STUDY. (Eng.) Boecker, W. (Inst. Pathology, Univ. Hamburg, Martinistrasse 52, D-2000 Hamburg 20, W. Germany); Lietz, H.; Delling, G. *Z Krebsforsch* 86 (1): 33-46; 1976.

Light and electron microscopy were used to correlate the histological and fine structural characteristics of an endodermal sinus tumor of the ovary of a 12-yr-old girl to clarify its possible histogenesis. The histological appearance was characterized by glandular-papillary structures and microcystic-reticular areas with inclusion of occasional endodermal sinuses. Immature and highly differentiated cells were distinguished by electron microscopy. The most differentiated cells in the tumor were regarded as neoplastic endoderm. The cells in mesenchymelike areas differed from those of solid and glandular parts in the degree of cytoplasmic differentiation but were otherwise believed to represent the same ''cell line.'' In addition to other possible cell functions, the cytoplasmic features of the differentiated cell indicate a protein synthesis and secretion. It is concluded that the endodermal sinus tumor originates from germ cells and differentiates into yolk sac endoderm. This endoderm, on the basis of microscopic evidence, can be differentiated clearly from a clear cell carcinoma.

2838 THE STEIN-LEVENTHAL SYNDROME AND COINCIDENTAL OVARIAN TUMORS. (Eng.) Babaknia, A. (Johns Hopkins Univ. Sch. Medicine, Baltimore, MD); Calfopoulos, P.; Jones, H. W., Jr. *Obstet Gynecol* 47(2): 223-224; 1976.

The findings from 181 cases of Stein-Leventhal syndrome over a 25-yr period were reviewed with attention to ovarian tumors and other associated pelvic lesions. A total of 28 ovarian tumors were found, of which the most common types were dermoid cyst, papillary fibroma and papilloma, cystic granulosa cell tumor, pseudomucinous cystadenoma, papillary cystadenoma, and hilar cell tumor. The most common incidental findings were myoma of the uterus, pelvic adhesions, endometrial hyperplasia, paraovarian cyst, and adenomyosis. These and others occurred in 36 (20%) of the patients. It is concluded that in planning treatment for this syndrome, the possibility of an ovarian tumor should be considered.

2839 FEMALE ADNEXAL TUMOR OF PROBABLE WOLFFIAN ORIGIN: EVIDENCE FOR A LOW GRADE MALIGNANCY. (Eng.) Taxy, J. B. (Northwestern Univ. Medical Sch., Chicago, IL); Battifora, H. *Cancer* 37(5): 2349-2354; 1976.

The case of a 41-yr-old woman with a broad ligament tumor with the histologic features of a female adnexal tumor of probable Wolfian origin is reported. Although previous cases were thought to be benign, hepatic metastasis occurred 6 yr after resection of the primary tumor. Light and electron microscopic studies revealed, in contrast to other tumors, no polarization of the cytoplasmic organelles, an absence of stereocilia, and microvilli generally confined to the network of intercellular spaces. Golgi area and free ribosomes were prevalent, but rough endoplasmic reticulum, secretory vacuoles, and mitochondria were sparse. Although its ultrastructural features do not correspond to those of normal or fetal mesonephric tissues, the tumor is probably of Wolffian origin.

2840 BENIGN VAGINAL RHABDOMYOMA: A LIGHT AND ELECTRON MICROSCOPIC STUDY. (Eng.) Gold, J. H. (Univ. Hosp., Minneapolis, MN 55455); Bossen, E. H. *Cancer* 37(5): 2283-2294; 1976.

A benign vaginal rhabdomyoma from a 49-yr-old woman was examined by light and electron microscopy. The unusual features were attachment plaques between cells enclosed within a common basement membrane, cytoplasmic bodies, and peripheral couplings. The ultrastructure was similar to that of an adult rhabdomyoma except for its lack of membrane-bound paracrystalline inclusions, tubular arrays in sarcoplasmic reticulum, and osmophilic inclusions in mitochondria. The Z-band material present did not form the ''rod'' inclusions found in typical adult rhabdomyosarcomas. Literature cases are reviewed and divided into adult and fetal types based on light microscopic findings.

2841 VAGINAL ADENOSIS AND DYSPLASIA AFTER ADMINISTRATION OF DIETHYLSTILBESTROL DURING UTERINE LIFE. (Fre.) Henry-Suchet, J. (Centre Hospitalier Universitaire Saint-Antoine, 184, rue du fg Saint-Antoine, 75571 Paris Cedex 12, France); Seneze, J.; Merger, R.; de Brux, J. *Ann Pediatr* 22(11): 835-839; 1975.

The literature concerning vaginal adenosis that resulted from treatment of pregnant women with diethylstilbestrol is reviewed. Three case histories (20, 17.5, and 21 yr) are also presented. It is recommended that all prepubescent and pubescent females whose mothers received diethylstilbestrol treatment be gynecologically followed. Recommended treatment is either surgical intervention or the placement of a 2 x/day capsule containing 20 mg of natural progesterone. This new latter treatment will, without interfering with the menstrual cycle, replace the glandular epithelium with normal malpighian epithelium within 2 yr.

2842 A MALIGNANT TUMOR OF THE VAGINA RESEMBLING SYNOVIAL SARCOMA: A LIGHT AND ELECTRON MICROSCOPIC STUDY. (Eng.) Okagaki, T. (Box 131, Mayo Memorial Building, Minneapolis, MN 55455); Ishida, T.; Hilgers, R. D. *Cancer* 37(5): 2306-2320; 1976.

A malignant tumor of the upper lateral vaginal mucosa in a 24-yr-old white woman was studied by light and electron microscopy. The main part of the tumor was epitheloid, and formed a tubular or acinar pattern. At the periphery, the tumor cells were more spindly and formed sheets resembling fibrosarcoma. The tumor cells contained occasional material positive for mucicarmine, alcian blue, and PAS stains; nests of the cells forming acini were surrounded by reticulin fibers. The tumor ultrastructure consisted of a single type of cell with large cytoplasmic vacuoles and occasional perinuclear intracytoplasmic filaments; slender long microvilli projected into the lumina. Basal laminae were occasionally present around the acini. The tumor probably was of mesenchymal origin, closely related to synovial sarcoma.

2843 GONADOBLASTOMA: ULTRASTRUCTURAL EVIDENCE FOR TESTICULAR ORIGIN. (Eng.) Ishida, T. (Univ. Minnesota Medical Sch., Minneapolis, MN); Tagatz, G. E.; Okagaki, T. *Cancer* 37(4): 1770-1781; 1976.

A gonadoblastoma arising in the dysgenetic gonad of a virilized 17-yr-old Caucasian with a female phenotype and with a 45,XO/46,X-ring-Y genotype was studied by light microscopic histochemistry, electron microscopy, and ultrastructural histochemistry. The gonadoblastoma was composed of nests of cells containing large germ cells and small ''granulosa-Sertoli-like cells,'' and stromal tissue containing ''Leydig-like cells.'' The germ cells were identical to those found in normal fetal gonads and in germ cell tumors. Charcot-Bottcher crystalloids present in the granulosa-Sertoli-like cells strongly suggested that they were, in fact, Sertoli cells. Multilayered basal laminae located in the periphery of the tumor nests and in ''hyaline bodies'' were identical to those surrounding the seminiferous tubules of the adult testis. The Leydig-like cells present in the stroma contained occasional dense bodies and crystalloids, which characterize the Leydig cells of the fetal testis. Δ^5-3β-hydroxysteroid dehydrogenase activity was demonstrated in the periphery of lipid droplets and lysosome-like dense bodies of the Leydig cells, and in some Sertoli cells. The findings support the theory that gonadoblastoma arises in a dysgenetic testis rather than in a dysgenetic ovary.

2844 MUTAGENIZED CLONES OF A PLURIPOTENT TERATOMA CELL LINE: VARIANTS WITH DECREASED DIFFERENTIATION OR TUMOUR-FORMATION ABILITY. (Eng.) Boon, T.; Kellermann, O.; Mathy, E.; Gaillard, J. A. In: *Teratomas and Differentiation.* Edited by M. I. Sherman and D. Solter. (New York: Academic Press, Inc.): pp. 161-166; 1975.

Clones of cells derived from pluripotent PCC4.azal teratoma cells after mutagenesis with 3 μg/ml of N-methyl-N-nitro-N-nitrosoguanidine were tested for their ability to produce differentiated tumors after the injection of 5×10^5 cells into syngeneic strain 129 mice. Of 65 clones analyzed, 5 (termed dif-) had a significantly reduced ability to form differentiated tumors and 12 (termed tum-) had a reduced ability to produce any tumors. As an example of the properties of a dif- clone, of 22 tumors formed by one clone, 18 were completely undifferentiated, 2 contained derivatives from one or two germ layers, and 2 contained derivatives of all three germ layers. On the other hand, of 23 PCC4.- azal tumors analyzed, none were undifferentiated, 2 contained derivatives of only one or two germ layers, and 21 contained ectodermal, mesodermal, and endodermal derivatives. When the host mice were irradiated prior to injection of the cells, tum- clones produced progressive tumors every time that grew as fast as the PCC4.azal tumors. It therefore appears that the tum- phenotype is due to an increased vulnerability of the growing cells to the defenses of the host. Such tum- variants and their tum control, PCC4.azal cells, offer a good system to study the interaction between cancerous cells and host rejection mechanisms. (6 references)

2845 PRIMARY CHORIOCARCINOMA OF THE MEDIASTINUM IN A MALE. (Fre.) Weiss, A.-M. (Hopital Beaujon, 100, bd General-Leclerc, 92110 Clichy, France); Frey, N.; Andreassian, B.; Potet, F. *Sem Hop Paris* 52(20): 1235-1238; 1976.

The clinical, radiological, and histopathological aspects of a rare case of primary choriocarcinoma of the mediastinum in a 19-yr-old male are presented, and the literature is reviewed. The patient was hospitalized initially for a pleural effusion of sudden onset. Physical examination revealed a malnourished male with bilateral gynecomastia and testicular atrophy. A tentative diagnosis of choriocarcinoma based on pathological levels of urinary chorionic gonoadotrophin was confirmed by exploratory thoracotomy. A large, multilobulated, necrotic, and hemorrhagic mass was removed from the mediastinal area. Complete excision was not possible because of adhesions to the thoracic and pulmonary arteries. Karyotyping of tumor cells revealed the presence of a Y chromosome and the absence of cells with an XX chromosome population. The patient died of massive metastasis on the 20th postoperative day. A precise histological study of the testes gave no evidence of tumor cells and confirmed the absence of spermatogenesis. Cases of suspected extragenital primary choriocarcinoma in the male should be confirmed by systematic histological examination of the testicular tissue. (16 references)

2846 ''ENDOMETRIAL'' ADENOCARCINOMA OF THE PROSTATE: A DISTINCTIVE TUMOR OF PROBABLE PROSTATIC DUCT ORIGIN.

(Eng.) Zaloudek, C. (M. D. Anderson Hosp. and Tumor Inst., Houston, TX); Williams, J. W.; Kempson, R. L. *Cancer* 37(5): 2255-2262; 1976.

The case history of a 76-yr-old man with an endometrial adenocarcinoma of the prostate that was studied by light and electron microscopy and by histochemical techniques is reported. Strong acid phosphatase staining and the ultrastructural demonstration of multivacuolated, lipid, and lysosome-containing tumor cells suggest a prostatic ductal origin for this type of carcinoma despite the histologic similarity to carcinoma of the endometrium.

2847 ULTRASTRUCTURAL STUDIES OF HUMAN PROSTATIC NEOPLASIA.

(Eng.) Ohtsuki, Y. (Texas Medical Center, Houston, TX 77025); Seman, G.; Maruyama, K.; Bowen, J. M.; Johnson, D. E.; Dmochowski, L. *Cancer* 37(5): 2295-2305; 1976.

To determine the possible viral etiology of human prostatic neoplasia, 42 specimens of the disease (32 carcinomas, 8 benign hyperplasia, and 2 bladder tumors infiltrating prostatic tissue) and 15 tissue cultures derived from prostatic neoplasia were examined by electron microscopy. Intracisternal viruslike particles, 150-200 nm in diameter and budding, were found in the epithelial cells of four carcinomas. In some of these particles, an electron-dense central core or two concentric layers were discernible. Particles resembling type C virus particles, 90-130 nm in diameter, were also observed in intracytoplasmic vacuoles in five cases of carcinomas and in one case of benign prostate hyperplasia. Virus particles were not found in any of the tissue culture specimens. Further studies are required to determine the nature of these particles and their relationship to the origin of human prostatic neoplasia. There were also intranuclear mitochondria, multilayered nuclear inclusions, bundles of intranuclear fibrils, intracytoplasmic tubules, extracellular tubulofilamentous structures, and cilia in both the benign hyperplasias and the carcinomas.

2848 HUMAN PROSTATIC ADENOMA AND CARCINOMA. TRANSPLANTATION OF CULTURED CELLS AND PRIMARY TISSUE FRAGMENTS IN ''NUDE'' MICE.

(Eng.) Okada, K. (Urologische Universitaetsklinik, 87 Wuerzburg, W. Germany); Schroeder, F. H.; Jellinghaus, W.; Wullstein, H. K.; Heinemeyer, H. M. *Invest Urol* 13(6): 395-403; 1976.

Tissues and cells from permanent cell lines of human prostatic carcinomas and adenomas were transplanted into the subcutaneous space of nude mice for a total of 208 transplantations. Transplantation of single tissue pieces of about 50 mg from the tumor provided the best results. This technique allowed the recovery of 5 of 10 types of carcinoma transplanted into 46 nude mice. Carcinoma was found in 7 of 25 nude mice that were castrated and treated with testosterone but only once in 21 transplantations into castrates without testosterone substitution. This suggests that heterotransplants of human prostatic carcinoma tissue can be established more easily in testosteronized animals. The histologic picture of the adenoma transplants was strikingly similar to the original tumors. Secretory activity was found in some acini, squamous metaplasia in others. Heterotransplantation of cells from permanent cell lines resulted in fast-growing carcinomas without similarity to the original tumors. Dependence on hormones for growth of EB 33 and MA 160 cells in nude mice was not shown.

2849 CYTOLOGICAL AND CYTOGENETICAL STUDIES ON BRAIN TUMORS. IV. NO EVIDENCE FOR A TRANSLOCATION IN 22-MONOSOMIC MENINGIOMAS.

(Eng.) Zankl, H. (Dept. Human Genetics, Univ. Saarland, Homburg, W. Germany); Weiss, A. F.; Zang, K. D. *Humangenetik* 30(4): 343-348; 1975.

Twelve human meningiomas were studied to determine whether they actually have a monosomy 22 or whether they have a translocation of all or part of chromosome 22. By conventional karyotyping and analysis of banding patterns, the tumors were divided into four groups. Four tumors showed exclusive loss of one chromosome 22, three showed loss of other chromosomes in addition to chromosome 22, two showed trisomies of other chromosomes in addition to the monosomy 22, and three showed structural aberrations in addition to the monosomy 22 and loss of other chromosomes. The missing or additional chromosomes in all tumors of the first three groups could be identified by their banding patterns. The lack of additional bands indicated that no chromosome 22 material was translocated. Two of the tumors in the fourth group had marker chromosomes that showed such complex compositions that the existence of a 22 translocation could not be excluded, although there was no positive sign of translocation. It is concluded that meningioma cells show a regular loss of a definitive part of their genome.

2850 METASTATIC CARCINOMA IN MENINGIOMA. (Eng.)

Gyori, E. (3200 Kirk St., Miami, FL 33133). *South Med J* 69(4): 514-517; 1976.

A case history of a 69-yr-old woman with adenocarcinoma of the lung metastatic to two of three parasagittal meningiomas is presented. Whether the metastasis was incidental or part of a massive cerebral involvement is not known. The amount of collagen fibers may have played a role in the metastasis because the smallest meningioma contained the least collagen and the larger metastatic ones contained the most. Also, the scarcity of ''resistant'' psammoma bodies may have attracted the carcinoma to the meningiomas.

2851 MELANIN AS A COMPONENT OF CEREBRAL GLIOMAS: THE MELANOTIC CEREBRAL EPENDYMOMA. (Eng.)

McCloskey, J. J. (Univ. Kentucky Medical Center, Lexington, KY 40506); Parker, J. C.; Brooks, W. H.; Blacker, H. M. *Cancer* 37(5): 2373-2379; 1976.

The case history of a 30-yr-old woman with an intracerebral melanin-containing glial tumor with features of a well-differentiated ependymoma is presented. Light microscopy revealed a moderately cellular tumor with a glial fiber matrix and scattered blood vessels. Melanin was distributed throughout the cytoplasm of the cells; the nuclei of the cells were generally oval. Electron microscopy revealed dark and light dense intracytoplasmic bodies consistent with melanosomes; no premelanosomes were evident. The presence of melanin appears to be a multipotential expression of neuroepithelial cells originating from the primitive neural tube, which is generally last in the process of normal maturation.

2852 HYPERPARATHYROIDISM AND CANCER. (Eng.)

Farr, H. W. (Memorial Sloan-Kettering Cancer Center, New York, NY). *CA* 26(2): 66-74; 1976.

The coincidence of hyperparathyroidism and cancer presents a critical problem in the differential diagnosis of hypercalcemia. Among 100 patients with proved parathyroid tumors, 75% were female and the median age at diagnosis was 55 yr. In 34/100, cancer was found before, during, or after removal of a benign parathyroid tumor. Five patients had coincidental mixed papillary thyroid carcinoma. Other common sites in these 34 patients were stomach (12), breast (19), skin (8), and colon (6). One-third of the series had benign unrelated tumors, and one-third had benign tumors associated with hyperparathyroidism. Early in the series, parathyroid tumors were 3-5 cm at diagnosis and extensive bone and renal disease were present, but improved diagnostic methods have been associated with smaller tumors at diagnosis. Hypercalcemia, hypophosphatemia, and hypercalciuria are common findings. The presence of hypercalcemia and elevated parathyroid hormone should be considered indicative of primary hyperparathyroidism until proved otherwise. Surgical exploration should be part of the diagnostic workup, as it is safe and 95% successful in correcting the problem. The diagnostic investigation and treatment of these patients is still unduly prolonged. Progressive renal damage and reduced lifespan have been well documented.

2853 GALACTORRHOEA, HYPERPROLACTINAEMIA AND PITUITARY TUMORS IN THE FEMALE. (Eng.)

Nader, S. (Royal Postgraduate Medical Sch., London W12 OHS, England); Mashiter, K.; Doyle, F. H.; Joplin, G. F. *Clin Endocrinol (Oxf)* 5(3): 245-251; 1976.

Sixty-two women with pituitary tumors but neither acromegaly nor Cushing's disease were analyzed with respect to mode of presentation. Thirty-six (58%) presented amenorrhea and galactorrhea; another 15 (24%) presented only amenorrhea. Pretreatment prolactin levels were available in 34 of these patients. In 27 (79%), hyperprolactinemia was found; this occurred in the absence of a history of galactorrhea in 4/10. In addition, 12 pituitary tumors were found in 25 unselected women with unexplained galactorrhea; amenorrhea was a feature of all. It is concluded that the incidence of hyperprolactinemia in women with apparently functionless pituitary tumors is higher than previously estimated. Serum prolactin should be assayed in all patients with such tumors even in the absence of a history of galactorrhea. All patients with unexplained galactorrhea should undergo careful radiological investigation for a possible pituitary tumor.

2854 ASSOCIATED RENAL POLYCYSTOSIS AND ADRENAL GLAND ADENOMA COMPLICATED BY RENAL HEMORRHAGE: REPORT OF TWO CASES. (Fre.)

Delavierre, P. (Laboratoire d'Anatomie Pathologique, Hopital de Vaugirard, 389, rue de Vaugirard, 75015 Paris, France); Vayre, P. *Sem Hop Paris* 52(20): 1223-1224; 1976.

The rare association of polycystosis of the kidney and adrenal gland adenoma was observed in two cases. The first was a 68-yr-old woman who was hospitalized because of renal pain. Hematuria was not present. Urography suggested a renal tumor, and the left kidney was removed because of the presence of a large hematoma. Pathological examination revealed renal polycystosis. There was an adenomatous nodule in the adrenal gland that was replaced in part by hematoma. The second case was a 30-yr-old man who was hospitalized in a deep coma. He had been severely hypertensive as a result of bilateral renal polycystosis. At autopsy, both kidneys were found to be greatly enlarged, with renal tissue largely replaced by blood-filled cysts. A well-circumscribed adenoma was found in the right adrenal gland. Hemorrhage of cysts or kidney tissue is frequently observed in renal polycystosis. In the first case, corticosteroid medication for arthritis may have caused the hemorrhage. (2 references)

2855 A TESTOSTERONE-PRODUCING ADRENAL CORTICAL ADENOMA IN AN ELDERLY WOMAN. (Eng.)

Larson, B. A. (Center for Endocrinology, Metabolism, and Nutrition, Northwestern Univ., Chicago, IL 60611); Vanderlaan, W. P.; Judd, H. L.; McCullough, D. L. *J Clin Endocrinol Metab* 42(5): 882-887; 1976.

The case history is presented of a 76-yr-old woman with virilization who had menopausal levels of circulating luteinizing hormone (LH) and follicle-stimulating hormone (FSH) and a markedly elevated concentration of plasma testosterone (9,130 pg/ml) into the range for adult men. Plasma cortisol and androstenedione levels and urinary 17-ketosteroid secretion were normal. Ethinyl estradiol treatment suppressed plasma testosterone, LH, and FSH levels into the normal range for premenopausal women, but the testosterone concentration was unaffected by the administration of dexamethasone or ACTH. Retrograde venous sampling and angiography localized a right adrenal adenoma preoperatively. Following adrenalectomy, there was a prompt fall in testosterone, but there was no change in the LH concentration. Thus, this patient had an adrenal adenoma that secreted only' testosterone and appeared to be gonadotropin-responsive. Testosterone levels in the adult male range failed to suppress the gonadotropins. Virilization occured after the patient had undergone surgically induced menopause. Whether this episode and the subsequent virilization share a common etiology is unclear; the development and secretory capacity, however, of the right adrenal tumor may have been abetted by the high circulating levels of postmenopausal gonadotropins. The low urinary 17-ketosteroid levels with high plasma testosterone levels emphasize the value of plasma testosterone as an indicator of virilization. The pattern of hormonal response to stimulation and suppression suggests that the tumor may have had a gonadal origin. Because one ovary had not been located at surgery 40 yr earlier and because of the tumor's apparent responsiveness to gonadotropins and unresponsiveness to ACTH, the exact location of the tumor was unclear after endocrine evaluation. The use of retrograde venous sampling of ovarian and adrenal veins may effectively locate the source of excess androgens in virilized patients.

2856 SPINDLE CELL CARCINOMA: ULTRASTRUCTURAL EVIDENCE OF SQUAMOUS ORIGIN AND COLLAGEN PRODUCTION BY THE TUMOR CELLS. (Eng.) Battifora, H. (Northwestern Memorial Hosp., 250 East Superior St., Chicago, IL 60611). *Cancer* 37(5): 2275-2282; 1976.

Transmission electron microscopy was used to study spindle cell carcinoma (SCC) of the esophagus and the skin in two men aged 57 and 62 yr, respectively. The tumor cells were closely associated with collagen fibrils and had abundant rough endoplasmic reticulum with dilated cisternae. Except for their irregular shape and atypical nuclei they resembled actively synthesizing fibroblasts. Some cells also contained numerous tonofibrils and occasional well-developed desmosomes. A gradual transition to typical squamous cells was noted in the skin tumor. These findings suggest that the pseudosarcomatous component of SCC originates from the mesenchymal metaplasia of squamous cells and that collagen is produced by these metaplastic cells.

2857 DEMONSTRATION OF 3-METHOXYTYROSINE IN THE URINE OF MELANOMA PATIENTS. (Eng.) Agrup, G. (Dept. Dermatology, Lasarettet, S-221 85, Lund, Sweden); Bengtsson, M.; Falck, B.; Persson, K.; Rorsman, H.; Rosengren, A.-M.; Rosengren, E. *Acta Derm Venerol (Stockh)* 56(1): 27-31; 1976.

3-Methoxytyrosine, a metabolite of dopa [3-(3,4-dihydroxyphenyl)-L-alanine], was demonstrated in the urine of two patients, aged 46 and 64 yr, with melanoma metastases. The amounts excreted were 4.0 and 8.5 mg/24 hr, respectively. No 3-methoxytyrosine was identified in the urine of two healthy controls. These findings suggest that the determination of this compound in the urine of patients with melanoma may prove important in investigations of the catechol metabolism of these patients.

2858 TYROSINASES OF NEVUS CELL NEVI AND MALIGNANT MELANOMAS IN HUMANS. (Fre.) Voulot, C. (Laboratoire de Biologie, INSA, F 69621 Villeurbanne, France); Ortonne, J.-P. *Ann Dermatol Syphiligr (Paris)* 102(5-6): 547-551; 1975.

Nine samples of nevocytic nevi (seven junctional nevi, one cerebriform nevus of the scalp, and one sparsely pigmented verruciform mole) were studied by gel electrophoresis with an L-dopa substrate, and compared to four malignant melanomas (three primary and one lymph node metastasis). Each of the heavily pigmented malformations had two highly mobile dopamelanin bands that were linked to melanin synthesis. These bands were not present in the sparsely pigmented nevus, in the controls, or in the achronic portions of the melanomas. All of the samples also had two weakly mobile bands not linked to melanin synthesis that may have been induced by tyrosinase activity.

2859 INFANTILE DIGITAL FIBROMATOSIS. (Fre.) Bonerandi, J. J. (Hotel-Dieu, Place Daviel, F 13324 Marseille Cedex 1, France); Follana, J.; Migozzi, B.; Temime, P. *Ann Dermatol Syphiligr (Paris)* 103(2): 161-168; 1976.

The literature on 26 cases of infantile digital fibromatosis, also called neurofibrosarcoma, and recurring digital fibrous tumors of the infant, is reviewed, and one case is presented. Nodules that were projecting, sessile, and varying in size from 8-10 mm to pinheads were first observed on the toes and fingers of an otherwise normal female infant when she was 5 1/2 mo old. The nodules were red, slightly translucent, and surrounded by normal skin. X-rays of the feet did not show any lesions of the bone. Nodules responded to cryotherapy. Optical microscopy revealed proliferation of collagenous tissue containing fusiform or star-shaped fibroblasts with irregular but not atypical nuclei. Elastic tissue was absent and capillaries were dilated. Round, dense, intracytoplasmic inclusions, stained red by eosin, were seen in some of the fibroblasts. Electron microscope studies defined these inclusion bodies clearly as granulofilamentous and detected smaller, less dense inclusions as well as highly osmiophilic, lamellar, intracytoplasmic ones. Also observed were intranuclear, granulofilamentous inclusions surrounded by a clear halo. A viral etiology for the infantile fibromatosis has been suggested by several studies; however, a virus has yet to be isolated from the fibromatous nodules.

2860 PLASMACYTOMA ARISING IN A PACEMAKER POCKET. (Eng.) Hamaker, W. R. (Letterman Army Medical Center, Presidio of San Francisco, CA 94129); Lindell, M. E.; Gomez, A. C. *Ann Thorac Surg* 21(4): 354-356; 1976.

The case of a 48-yr-old man who developed a plasmacytoma in the subcutaneous pocket of a titanium-covered pulse generator that had been inserted 16 mo previously is presented. The tumor presented a cutaneous manifestation of myelomatosis. Despite substantial reduction of the tumor as a result of cobalt therapy, the patient died of progressive multiple myeloma and sepsis. The tumor is thought to be caused by thronic irritation, electro-chemimcal stimulation, or chemotaxis. The presence of a pacemaker may affect the detection and treatment of tumors arising in the region of pulse genearator pockets.

2861 ULTRASTRUCTURE OF OSTEOID OSTEOMA. (Eng.) Steiner, G. C. (Mount Sinai Sch. Medicine, City Univ. New York, NY). *Hum Pathol* 7(3): 309-325; 1976.

The ultrastructure in five cases (four males 21, 9, 22, and 18 yr; one woman 68 yr) of osteoid osteoma is described (and, below, compared to the ultrastructure of osteoblastoma). The osteoblasts, the predominant cells, generally had a morphology similar to that of normal osteoblasts, with a few differences. They contained irregular indented nuclei, glycogen particles, abundant fine intracytoplasmic fibrils, and rare iron-containing lysosomes. In several osteoblasts in two of the cases there were atypical mitochondria with a lobulated or ''honeycomb'' appearance. These mitochondria were also observed in two osteoclasts, which otherwise resembled normal osteoclasts. Other cells present in osteoid osteoma included osteoprogenitor cells resembling Scott type A and B cells and cells in transitional stages of differentiation. The osteoblasts most likely originated from Scott type A cells or preosteoblasts. The areas of mineralized matrix conformed to the morphology of coarse woven bone. Varying amounts of osteoid were noted. In two cases the osteoid contained, in addition to collagen, fine granular material, probably polysaccharides. No nerve fibers were identified in the material studied. It is thought that osteoid osteoma is a benign neoplastic lesion. Osteoblastoma cells had basically the same morphology as the cells in osteoid osteoma, including the atypical mitochondria. These ultrastructural observations support the idea that osteoid osteoma and osteoblastoma are closely related lesions.

2862 HIDROACANTHOMA SIMPLEX: AN ULTRASTRUCTURAL STUDY AND COMPARISON WITH ECCRINE POROMA. (Eng.) Oka, K. (Nagaoka Red Cross Hosp., 2-6-1 Nissekicho, Nagaoka, Japan); Morohashi, M.; Nitto, H. *J Dermatol* 2(2): 69-78; 1975.

The case history is presented of a 76-yr-old woman with a rare hidroacanthoma simplex tumor on the knee. It appeared as a Bowen's diseaselike lesion and was confirmed by clinical and histological examinations. Ultrastructurally, the tumor cells revealed a structure similar to that of eccrine poroma in that they had numerous glycogen granules and the ring-shaped distribution of tonofilaments, but unlike eccrine poroma they had no intracellular cavity formation and their tonofilaments and desmosomes were underdeveloped. These findings indicate that hidroacanthoma simplex is related to eccrine poroma, but is not identical to it nor an intraepidermal form of it.

2863 CARCINOSARCOMA OF THE ESOPHAGUS: STUDY OF ONE CASE AND LITERATURE REVIEW. (Fre.) Capron, J.-P. (Centre Hospitalier Universitaire, F 59000 Lille, France); Poissonnier, P.; Liber, F.; Gosselin, B.; Laget, J.-P. *Arch Fr Mal App Dig* 64(6): 529-534; 1975.

The literature on 50 cases of carcinosarcoma of the esophagus is reviewed, and a case in a 44-yr-old man is presented. Barium radiography and esophagoscopy revealed a 7 × 4 cm pedunculated, lobulated, polypoid mass obstructing the middle third of the esophagus. Biopsy suggested an epidermoid carcinoma. A partial esophagectomy followed by a terminal-lateral esophagogastrostomy was performed. Pathologically, the tumor appeared to be a poorly differentiated epidermoid carcinoma; however, at the center there was a fibroblastic-type sarcoma. Small islets of carcinomatous tissue were observed in the sarcoma. The patient remained well for 10 mo, but succumbed within the year to bone marrow aplasia secondary to sarcomatous invasion. The histogenesis of the carcinosarcoma is controversial. The combined tumor could be a result of: (a) a ''collision'' of two independently developing tumors; (b) a single cell of origin that differentiates into two types of tumor cells; (c) sarcomatous cells deriving from metaplastic carcinomatous cells. The similarity and possible identity of the carcinosarcoma and the pseudosarcoma are also considered. (43 references)

2864 ONCOCYTIC LESIONS OF THE UPPER RESPIRATORY TRACT. (Eng.) Busuttil, A. (Ear, Nose and Throat Hosp., Glasgow, Scotland). *J Laryngol Otol* 90(3): 277-288; 1976.

The histogenesis of the oncocyte and its relationship to carcinoid lesions and to adenolymphomas of the salivary glands are considered. During the period 1960-1974, more than 2,000 laryngeal biopsies were examined and 19 of 36 diagnosed laryngeal cysts had linings which were either totally or partially composed of oncocytes. Another cyst which was also lined by oncocytes was removed from the nasopharynx. All the oncocytic cysts removed were from patients over 50 yr old (5 male, 14 female). The oncocytic cysts were often found to be multicentric when examined histologically. The lining consisted of one or more layers of oncocytic cells. In five oncocytic cysts there were prominent papillary epithelial escrescences with a vascular core projecting into the cyst, giving the appearance of papillary cystadenomas. Inspissated mucus, muciphages, and acute and chronic inflammatory cells were in the lumen and surrounding connective tissue. Spaces within the oncocytic lining simulated intra-epithelial lumina. The surface squamous epithelial lining overlying the cysts showed localized hyperplasia and acanthosis. Two case histories are presented, one of a 76-yr-old male who complained of a 7-wk history of blockage of the right nasal passage, mild epistaxis, watery rhinorrhea, and a persistent clicking in the right ear. A 0.3-cm diameter rounded smooth lesion occluded the torus tubarius. Excision biopsy showed a simple cyst entirely lined by oncocytes and surrounded by normal nasopharyngeal lymphoid tissue. The second case involved a 63-yr-old teacher who presented with hoarseness of a few months' duration, persistent nasal obstruction and watery discharge. A cyst was seen on the vocal cord which was shown to be an oncocytic-lined cyst. The nasal mucous glands within the myxoid stroma of the polyp showed prominent oncocytic change. Ten years later, she again developed hoarseness. In biopsy the laryngeal glands of both ventricles showed marked oncocytic changes. The present consensus is that conversion to an oncocytic appearance is the result of physiological changes related to functional exhaustion. One suggested explanation for the oncocyte's appearance is that as the cell ages and the main line of genetic information from the nucleus starts to falter, the mitochondrial DNA takes over and secondary hyperplasia of the mitochondria occurs.

2865 SCLERODERMA AND CARCINOMA OF THE LUNG.

(Eng.) Twersky, J. (State Univ. New York Downstate Medical Center, Brooklyn, NY 11203); Twersky, N.; Lehr, C. *Clin Radiol* 27(2): 203-209; 1976.

Two cases of scleroderma and carcinoma of the lung in a 44- and a 61-yr-old woman are reported. The 16 previously reported cases in the English literature are reviewed and tabulated in an attempt to determine the pathological and clinical presentation of carcinoma of the lung superimposed on the sclerodermal lung. When patients with scleroderma develop a change in pulmonary symptoms, a superimposed cancer of the lung should be suspected. The relationship between interstitial pulmonary inflammatory disease and cancer may be explained in one of three possible ways: two different agents independently produce the inflammatory disease and the cancer; both inflammatory disease and cancer have a common cause; and, most likely, the atypical epithelium induced by one cause becomes susceptible to a carcinogen.

2866 LUNG CARCINOMA SUPERIMPOSED ON PULMONARY TUBERCULOSIS.

(Eng.) Ting, Y. M. (Wayne County General Hosp., Eloise, MI 48132); Church, W. R.; Ravikrishnan, K. P. *Radiology* 119: 307-312; 1976.

In a series of 450 patients with active-tuberculosis, 22 were shown to have coexisting carcinoma of the lung. There were 12 squamous cell tumors, 6 adenocarcinomas, 3 oat cell types, and 1 undifferentiated. Sputum cytology was positive in 9, negative in 11, and not determined in 2. Initial chest film suggested carcinoma in seven, five of whom had negative sputum cytology. Chest film findings in the remaining 15 were interpreted as reactivation of pulmonary tuberculosis or pneumonia. Of these, eight had positive cytology and six had negative cytology. Suspicious x-ray findings are reviewed, and the importance of sputum cytology is stressed.

2867 LYMPHANGIOMYOMATOSIS: MORPHOGENETIC STUDY AND ULTRASTRUCTURAL CONFIRMATION OF THE HISTOGENESIS OF THE LUNG LESION.

(Eng.) Vazquez, J. J. (Dept. Pathology, Univ. Navarra, Pamplona, Spain); Fernandez-Cuervo, L.; Fidalgo, B. *Cancer* 37(5): 2321-2328; 1976.

Two months after surgical intervention for a retroperitoneal lymphangiomyoma, a 34-yr-old woman developed a spontaneous right pneumothorax with a chylous pleural effusion. Roentgenograms showed that the lungs had developed a new honeycomb pattern. She was treated surgically and, at the same time a biopsy was performed. Sixteen months later she developed a left pneumothorax that necessitated additional surgery (a further biopsy was taken). She died of respiratory insufficiency 2 yr after the clinical onset of the symptoms. Electron microscopic study revealed the muscular nature of this condition. Light microscopic study of the serial sections showed that the lung lesion was due to proliferation of the muscle cells associated with the lymphatics surrounding the acini.

2868 BRONCHIAL CANCER WITH PARANEOPLASTIC HYPERCALCEMIA AND HYPERCALCITONINEMIA.

(Fre.) Akoun, G. (Hopital Tenon, 4, rue de la Chine, 75970 Paris Cedex 20, France); Huchon, G.; Marsac, J.; Isaac, R.; Chauveau, P.; Brocard, H. *Rev Fr Mal Respir* 4(2): 184-190; 1976.

A case of an epidermoid carcinoma of the bronchus accompanied by a hypercalcemic syndrome and elevated parathormone and calcitonin levels is presented. The patient, a 50-yr-old man, had a serum calcium level of 146 mg/l and a phosphorus level of 20 mg/l. Parathormone levels were 3.9 ng/ml (normal is under 1.5 ng/ml). Pulmonary artery and vein puncture during removal of tumor permitted measurement of arteriovenous differences in the levels of parathormone and calcitonin. Higher levels of calcitonin in the pulmonary artery than in the pulmonary vein indicated ectopic production by tumor tissue. After operation, calcium and phosphorus levels became normal. Recurrence of the tumor 3 mo after surgery was accompanied by elevated serum calcium, calcitonin, and parathormone levels. Serum prostaglandins also increased to above normal levels. The patient died, and widespread metastases were noted at autopsy. Hypercalcemia observed during the course of bronchial cancer can be due to a process other than bone lysis. In the case described, prostaglandins may have played a role in the hypercalcemic syndrome. (20 references)

2869 MICROINVASIVE CARCINOMA OF THE CERVIX.

(Eng.) Ruch, R. M. (20 S. Dudley, Memphis, TN 38103); Pitcock, J. A.; Ruch, W. A., Jr. *Am J Obstet Gynecol* 125(1): 87-92; 1976.

To evaluate the criteria of invasive depth, invasive pattern, and lymphatic involvement as indications for therapy for microinvasive carcinoma, 115 cases of this disease were reviewed. The 71 invading 1 mm or less were focal (96%) rather than confluent (4%) and were cured by simple hysterectomy (one vaginal vault recurrence). The 44 invading 2 to 5 mm showed a confluent pattern in 39%; a need for treating pelvic lymphatics was demonstrated by 1 with a positive hypogastric lymph node and by 3 pelvic recurrences. An additional 26 cases assessed as beyond microinvasive (6 to 10 mm in depth) included 5 of 16 without radical treatment who died from cancer, while all treated radically survived, including 5 with lymphatic involvement. The use of invasive depth as a criterion for evaluation of the prognosis of microinvasive carcinoma confirmed that lesions involving 1 mm or less can be successfully treated by simple hysterectomy; when microinvasion exceeds 1 mm, radical therapy must be considered. Microscopic patterns of invasion provided the best prognostic correlation.

2870 FAMILIAL CARCINOMA OF THE PANCREAS. (Eng.)

Friedman, J. M. (Veterans Admin. Hosp., Seattle, WA 98108); Fialkow, P. J. *Clin Genet* 9(5): 463-469; 1976.

A sibship is reported in which four brothers died of adenocarcinoma of the pancreas in the seventh or eighth decade of life. None of the patients had a history of pancreatitis, other malignant neoplasms, or exposure to a known pancreatic carcinogen. [A sister's daughter had carcinoma in situ of the cervix uteri (39 yr), a cousin had adenocarcinoma of the prostate (82 yr), another had adenocarcinoma of the ascending colon (female, 66 yr), and a third cousin (64 yr) had papillary carcinoma of the rectum and, 14 yr later, adenocarcinoma of the prostate.] This family apparently exhibits a genetic predisposition to pancreatic carcinoma late in life. The mode of inheritance and the relative importance of genetic and environmental factors in the development of these tumors are unclear.

2871 ABNORMAL TRYPTOPHAN METABOLISM IN A FAMILY WITH A HISTORY OF BLADDER CANCER. (Eng.)

Leklem, J. E. (Dept. Foods Nutrition, Oregon State Univ., Corvallis, OR 97331); Brown, R. R. *J Natl Cancer Inst* 56(6): 1101-1104; 1976.

Tryptophan metabolism was studied in five siblings of a family in which a sister and brother had bladder cancer. Urinary tryptophan metabolites after administration of 2 g L-tryptophan were measured in four sisters and one brother, all free of disease when studied. One sister with and two without histories of bladder cancer had significantly elevated levels of kynurenine, acetylkynurenine, and 3-hydroxykynurenine. Administration of 100 mg pyridoxine hydrochloride returned the tryptophan metabolism to normal in these three. One brother with and one sister without bladder cancer had normal metabolism. A repeat study 2 yr later confirmed the abnormal metabolism in the three sisters. Two sisters with abnormal tryptophan metabolism were given 200 mg L-kynurenine sulfate orally to bypass the effects of tryptophan oxygenase activity. Both excreted elevated levels of kynurenine and one excreted elevated levels of 3-hydroxykynurenine. The abnormalities in tryptophan metabolism are not implied to be due to the presence of bladder cancer, but are consistent with a defect, perhaps congenital, in the kynureninase enzyme. (22 references)

2872 EPITHELIAL TUMORS OF THE BLADDER. (Rus.)

Romanenko, A. M. (Kiev Inst. Food Hygiene, Ukrainian SSR Ministry Health, Kiev, USSR). *Arkh Patol* 38: 22-31; 1976.

The results of a study of 450 patients with epithelial tumors of the bladder are presented. The mucous membrane of the bladder taken far from the site of the tumor served as a control. The long-term results of surgical treatment were studied in 180 patients. The maturity of the neoplasm was determined by histological and histochemical methods. Twenty-two transitional-cell tumors of the bladder of different degrees of maturity were analyzed electron microscopically. The normal transitional epithelium exhibited high activity of the enzymes involved in respiration, in electron transport, and in the catabolic pathways of nucleic acid metabolism. In transitional-cell papilloma, transitional-cell highly differentiated cancer, and transitional-cell moderately differentiated cancer, this enzyme activity was inhibited markedly; however, the activity of the enzymes involved in the glycolysis chain and in the anabolic nucleic acid metabolism was increased. The heterogeneity of the transitional-cell neoplasms was also confirmed by the long-term results of surgical treatment. Poor prognosis is expected for the nondifferentiated cancer (only 58% of the patients lived 2-3 yr after surgical treatment), but prognosis for the transitional-cell papilloma is better (the 2- to 3-yr survival rate was 86%). (11 references)

2873 METHODS OF CHROMOSOME STUDY FOR HUMAN SOLID TUMOURS. (Eng.)

Slater, R. (Anthropobiologisch Lab., Amsterdam, The Netherlands). *Int Union Cancer Tech Rep Ser* 20: 63-68; 1975.

Procedures for preparing and staining chromosomes for the study of aberrations in solid tumors are described. The specimen should be from deep within the tumor mass and should be free of necrotic tissue. Some soft tumors form a suspension readily after a simple mincing with scissors, but others require use of disaggregating agents such as trypsin and collagenase, with the addition of DNase to prevent cell clumping. "The total time taken from the collection of the sample to making the chromosome preparations should be approximately three hours." To obtain metaphase chromosomes, the tumor cell suspension is treated with colcemid to arrest the cells at metaphase. Problems with chromosome spreading on the microscope slides may arise due to insufficiently clean slides, poor fixation and too great a cell concentration. Metaphase chromosomes obtained from human preparations are often of bad morphology and have a fuzzy appearance; short-term culture may yield better preparations more suitable for banding staining. Three methods of chromosome staining are considered: lacto-acetic orcein, a good overall morphological stain, and two banding techniques, ASG (acetic/saline/Giemsa) and ASG/trypsin. For optimum results with the two banding techniques, chromosome preparations should not be more than 3 days old. The two banding techniques give greater chromosome detail than the first stain and allow the recognition of individual chromosomes and changes, both numerical and structural, in the karyotype.

2874 METASTASIS FORMATION AFTER INTRAVENOUS TUMOR CELL INJECTION IN THROMBOCYTOPENIC RATS. (Eng.) Ivarsson, L. (Dept. Surgery I, Sahlgrenska sjukhuset, Univ. Goeteborg, Goeteborg, Sweden). *Eur Surg Res* 8(1): 51-60; 1976.

The effect of platelet reduction on the trauma- and dextran 1000 (0.75 ml/100 g, iv)-induced formation of metastases was studied in inbred Wistar rats injected iv with transplantable 20-methylcholanthrene-induced fibrosarcoma cells. The trauma was a bilateral femoral crush fracture, and platelet reduction was induced by the iv injection of 0.2 ml rat antiplatelet serum (APS). APS treatment alone significantly decreased the number of metastases and the total metastasis volume, whereas trauma and dextran 1000 alone had the opposite effect. When APS treatment was combined with trauma, the total metastasis volume was insignificantly decreased, the number of metastases tended to be significantly decreased, and the median metastasis volume was insignificantly increased. There were no significant differences between the effects of APS treatment alone and APS treatment combined with trauma, but the differences in total metastasis volume and number of metastases were highly significant when the effect of APS treatment plus trauma was compared with that of trauma alone. When APS treatment was combined with dextran 1000, the effect on metastasis was about the same as that of dextran 1000 alone. No extrapulmonary metastases were observed in any animals. The results indicate that platelets are important for the increased formation of metastases after trauma but not for the increased formation of metastases after dextran 1000 infusion. Thus, trauma and dextran 1000 stimulate metastasis formation by different mechanisms.

2875 INTRACYTOPLASMIC TUBORETICULAR STRUCTURES: PRESENCE IN A HUMAN LYMPHOSARCOMA. (Fre.) Reynes, M. (Hotel Dieu, 1 Place du Parvis Notre-Dame, 75181 Paris Cedex 04, France); Diebold, J. *Ann Anat Pathol (Paris)* 20(2): 149-158; 1975.

2876 JEJUNAL RETICULOSARCOMA ASSOCIATED WITH CELIAC DISEASE IN AN ADULT. (Fre.) Capron, J. P. (Centre Hospitalier Universitaire, 1, place Victor-Pauchet, F 80000 Amiens, France); Marti, R.; Delamarre, J. *Nouv Presse Med* 5(2): 80-82; 1976.

2877 HEPATIC ANGIOSARCOMA IN A VINYL CHLORIDE WORKER. (Eng.) Smith, P. M. (Llandough Hosp., B.P. Chemicals International Ltd., Penarth, Glamorgan, Wales); Williams, D. M. J.; Evans, D. M. D. *Bull NY Acad Med* 52(4): 447-452; 1976.

2878 A CASE OF LIVER ANGIOSARCOMA IN A WORKER EXPOSED TO MONOMERIC VINYL CHLORIDE. (Fre.) Ravier, E. (Societe des Usines chimiques Rhone-Poulenc, Usine de Rousillon 38, Roussillon, France); Diter, J. M.; Pialat, J. *Arch Mal Prof Med Trav Secur Soc* 36(3): 171-177; 1975.

2879 THE ASSOCIATION OF KAPOSI'S CUTANEOUS SARCOMA WITH MEDIASTINAL HODGKIN'S DISEASE. (Fre.) Beurey, J. (Hopital Fournier, 36, quai de la Bataille, F 54035 Nancy Cedex, France); Weber, M.; Mazet, J.; de Ren, G.; Vaillant, G. *Ann Dermatol Syphiligr (Paris)* 103(2): 151-159; 1976.

2880 HODGKIN'S DISEASE AND HYDANTOIN. (Fre.) Thibaut, G. (Clinique Medicale A, Nancy, France); Guerci, O.; Puchelle, J. C.; Lederlin, P.; Aymard, J. P.; Witz, F.; Weber, M. *Ann Med Nancy* 15: 29-30, 32; 1976.

2881 BIOLOGY OF THE HUMAN MALIGNANT LYMPHOMAS. III. INTRACRANIAL HETEROTRANSPLANTATION IN THE NUDE, ATHYMIC MOUSE. (Eng.) Epstein, A. L. (Stanford Univ. Sch. Medicine, Stanford, CA 94305); Herman, M. M.; Kim, H.; Dorfman, R. F.; Kaplan, H. S. *Cancer* 37(5): 2158-2176; 1976.

2882 AUTORADIOGRAPHIC STUDY OF THE RELATIONSHIPS BETWEEN THE ULTRASTRUCTURAL MORPHOLOGY OF LYMPHOID CELLS OF THE THYMUS AND THEIR FUNCTION IN THYMIC LYMPHOPOIESIS. THEIR ROLE IN RADIOGENIC LEUKEMOGENESIS (Abstract). (Fre.) Boniver, J. (Laboratoire d'Anatomie Pathologique, Universite de Liege, 1, rue des Bonnes-Villes, B-4000 Liege, Belgium); Courtoy, R.; Simar, L. J. *J Microscop Biol Cellul* 23(2): 32a-33a; 1975.

2883 CASE RECORDS OF THE MASSACHUSETTS GENERAL HOSPITAL: WEEKLY CLINICOPATHOLOGICAL EXERCISES. (Eng.) Scully, R. E. (Massachusetts General Hosp., Boston, MA); Galdabini, J. J.; McNeely, B. U. *N Engl J Med* 294(24): 1333-1338; 1976.

2884 COMMON TUMORS OF RETICULAR TISSUES IN MICE. (Eng.) Metcalf, D. (Walter and Eliza Hall Inst., Melbourne, Australia). *Int Union Cancer Tech Rep Ser* 20: 31-35; 1975.

2885 CAPILLARY MIGRATION OF THE CELLS OF THREE MURINE TUMOURS. (Eng.) Boranic, M. (Dept. Experimental Biology Medicine, Rudjer Boskovic Inst., 41001 Zagreb, P.O. Box 1016, Yugoslavia); Sabioncello, A.; Radacic, M.; Dekaris, D.; Veselic, B. *Biomed Express (Paris)* 25(1): 15-18; 1976.

2886 FINE STRUCTURE OF A ''POLYRIBOSOME-LAMELLA'' COMPLEX IN AN ABDOMINAL CHEMODECTOMA (Abstract). (Fre.) Sonsino, E. (Hopital Bichat, 170, boulevard Ney, 75018 Paris, France);

Nabarra, B.; Bocquet, L. *J Microscop Biol Cellul* 23(2): 79a; 1975.

2887 PRIMARY SQUAMOUS CELL CARCINOMA OF THE PAROTID GLAND. (Eng.) Batsakis, J. G. (Univ. Michigan, Ann Arbor, MI); McClatchey, K. D.; Johns, M.; Regazi, J. *Arch Otolaryngol* 102(6); 355-357; 1976.

2888 SEBACEOUS LYMPHADENOMA OF THE PAROTID GLAND. (Eng.) Baratz, M. (Tel Aviv Univ. Medical Sch., Tel Aviv, Israel); Loewenthal, M,; Rozin, M. *Arch Pathol Lab Med* 100(5): 269-270; 1976.

2889 COMPLEX TUMOR OF THE SUDORIFEROUS GLANDS IN THE SCALP. ASSOCIATION OF A SYRINGOCYSTADENOMA PAPILLIFERUM AND AN APOCRINE HIDROCYSTOMA. (Fre.) Vittori, F. (Hotel-Dieu, 1, place de l'Hopital, 69002 Lyon, France); Colomb, D. *Arch Anat Cytol Pathol* 24(1): 51-55; 1976.

2890 ADENOMAS AND CARCINOMAS OF THE CANINE AND FELINE THYROID. (Eng.) Leav, I. (Tufts Univ. Sch. Medicine, 136 Harrison Ave., Boston, MA 02111); Schiller, A. L.; Rijnberk, A.; Legg, M. A.; der Kinderen, P. J. *Am J Pathol* 83(1): 61-94; 1976.

2891 DISTRIBUTION OF GLANDS IN THE MUCOSA OF THE HARD PALATE AND ITS RELATION TO CARCINOMA. (Eng.) Reddy, C. R. R. M. (Andhra Medical Coll., Visakhapatnam-2, India); Venkatarathnam, G.; Kameswari, V. R. *J Oral Surg* 34(3): 232-236; 1976.

2892 METASTATIC HYPERNEPHROMA TO THE HEAD AND NECK. (Eng.) Schantz, J. C. (Milton S. Hershey Medical Center, Pennsylvania State Univ., Hersey, PA 17033); Miller, S. H.; Graham, W, P., III *J Surg Oncol* 8(2): 183-190; 1976.

2893 RETINOBLASTOMA OCCURRING IN FOUR SUCCESSIVE GENERATIONS. (Eng.) Migdal, C. (St. Bartholomew's Hosp., London, England). *Br J Ophthalmol* 60(2): 151-152; 1976.

2894 CARCINOMA ARISING IN THE AREA OF MEDIAN RHOMBOID GLOSSITIS. (Eng.) Burkes, E. J., Jr. (Univ. North Carolina Sch. Dentistry, Chapel Hill, NC); Lewis, J. R. *Oral Surg* 41(5): 649-652; 1976.

2895 SCHWANNOMA PRESENTING AS A NASAL POLYP. (Eng.) Kaufman, S. M. (Coral Gables Hosp., 3100 Douglas Road, P.O. Box 340610, Coral Gables, FL 33134); Conrad, L. P. *Laryngoscope* 86(4): 595-597; 1976.

2896 ADENOCARCINOMA OF THE PARA-NASAL SINUSES. (Eng.) Saunders, S. H. (Saint James' Hosp., London SW12 8HW, England); Ruff, T. *J Laryngol Otol* 90(2): 157-166; 1976.

2897 LEIOMYOMA ARISING ADJACENT TO A MAXILLARY TOOTH SOCKET: AN INTRAOSSEOUS LEIOMYOMA PRESENTING AS AN ODONTOGENIC LESION. (Eng.) Rhatigan, R. M. (Univ. Hosp. Jacksonville, 655 W. Eighth St., Jacksonville, FL 32209); Kim, Z. E. *South Med J* 69(4): 493-494; 1976.

2898 MICROSCOPIC CHARACTERISTICS OF THE ACOUSTIC TUMOR IN RELATIONSHIP OF ITS NERVE OF ORIGIN. (Eng.) Neely, J. G. (Dept. Otorhinolaryngology and Communicative Sciences, Baylor Coll. Medicine, Houston, TX 77030); Britton, B. H.; Greenberg, S. D. *Laryngoscope* 86(7): 984-991; 1976.

2899 STUDIES ON EXPERIMENTAL MALIGNANT NERVE SHEATH TUMORS MAINTAINED IN TISSUE AND ORGAN CULTURE SYSTEMS I. LIGHT MICROSCOPY OBSERVATIONS. (Eng.) Rubinstein, L. J. (Stanford Univ. Sch. Medicine, Stanford, CA 94305); Conley, F. K.; Herman, M. M. *Acta Neuropathol (Berl)* 34(4): 277-291; 1976.

2900 STUDIES ON EXPERIMENTAL MALIGNANT NERVE SHEATH TUMORS MAINTAINED IN TISSUE AND ORGAN CULTURE SYSTEMS. II. ELECTRON MICROSCOPY OBSERVATIONS. (Eng.) Conley, F. K. (Stanford Univ. Sch. Medicine, Stanford CA 94305); Rubinstein, L. J.; Spence, A. M. *Acta Neuropathol* 34(4): 293-310; 1976.

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2904 CYTOLOGIC CHANGES IN AN OVARIAN THECOMA (Letter to Editor). (Eng.) de Torres, E. F. (Instituto Nacional de Cancerologia, Ninos Heroes 151, Mexico 7, D.F.). *Acta Cytol* 20(2): 104-105; 1976.

2905 GENITAL LESIONS IN THE PEUTZ-JEGHERS SYNDROME: WITH REFERENCE TO ONE CASE. (Fre.) Lecomte, P. (Centre Hospitalier Universitaire Bretonneau, 2, boulevard Tonnelle, 37033 Tours Cedex, France); Lansac, J.; Berger, C. *Rev Fr Gynecol* 71(2): 107-111; 1976.

2906 EFFECT OF OOPHORECTOMY ON THE GROWTH RATE OF TRANSPLANTED DIMETHYLBENZANTHRACENE-INDUCED OVARIAN GRANULOSA CELL TUMORS IN MICE. (Eng.) Brandes, J. M. (Rambam Government Hosp., Technion-Israel Inst. Technology, Haifa, Israel); Katz, M.; Robinson, E. *Israel J Med Sci* 12(2): 164-167; 1976.

2907 SQUAMOUS CELL NEOPLASTIC CHANGE IN PATIENTS WITH VAGINAL ADENOSIS (Eng.) Fetherston, W. C. (Saint Mary's Hosp., 2320 N. Lake Drive, P.O. Box 503, Milwaukee, WI 53211). *Am J Obstet Gynecol* 124(6): 665-666; 1976.

2908 VAGINAL ADENOSIS AND DYSPLASIA AFTER ADMINISTRATION OF DIETHYLSTILBESTROL DURING UTERINE LIFE. (Fre.) Henry-Suchet, J. (Centre Hospitalier Universitaire Saint-Antoine, 184, rue du fg Saint-Antoine, 75571 Paris Cedex 12, France); Seneze, J.; Merger, R.; de Brux, J. *Ann Pediatr* [*part of Semaine des Hopitaux*] 51(43): 835-839; 1975.

2909 STILBESTROL-INDUCED MULLERIAN CERVIX-A COLPOSCOPIC STUDY. (Eng.) Baggish, M. S. (Mount Sinai Hosp. Hartford, CT); Robinson, D.; Passman, B. *Gynecol Oncol* 4(1): 20-32; 1976.

2910 AN ULTRASTRUCTURAL AND HISTOCHEMICAL STUDY OF THE MUCOUS MEMBRANE ADJACENT TO AND REMOTE FROM CARCINOMA OF THE COLON. (Eng.) Dawson, P. A. (Westminster Medical Sch., London, England); Filipe, M. I. *Cancer* 37(5): 2388-2398; 1976.

2911 PREDICTION OF EARLY RECURRENCE OF COLON CANCER (Proceedings Abstract). (Eng.) Bivins, B. A. (Albert C. Chandler Coll. Medicine, Lexington, KY 40506); Somes, G.; Kashmiri, R.; Meeker, W. R. *IRCS Med Sci (Suppl)* 3(10): 18; 1975.

2912 HYPERPLASTIC POLYPS OF THE COLON. (Fre.) Lesbros, F. (Hopital E.-Herriot, place d'Arsonval, 69003 Lyon, France); Maenhout, C.; Moulay, A.; Nemoz, C.; Site, J. *Arch Anat Cytol Pathol* 24(1): 31-36; 1976.

2913 INSULINOMAS IN CHILDREN. HISTOLOGICAL AND ULTRASTRUCTURAL STUDY OF TWO CASES. (Fre.) Casanova, P. (Groupe hospitalier de la Timone, Secteur universitaire, chemin de l'Armee d'Afrique, 13385 Marseille Cedex 4, France); Guidon, J.; Laffargue, P. *Ann Anat Pathol (Paris)* 20(5): 489-503; 1975.

2914 ULTRASTRUCTURAL STUDY OF THE INVASION OF THE LIVER AND STRIATED MUSCLES BY TRANSPLANTABLE TUMORS (Abstract). (Fre.) Babai, F. (Departement de Pathologie, Universite de Montreal, C.P. 6128, Montreal 101, Quebec, Canada). *J Microscop Biol Cellul* 23(2): 27a; 1975.

2915 FINE STRUCTURAL STUDY OF IN VITRO INTERACTIONS BETWEEN CANCER CELLS AND THE LIVER (Proceedings Abstract). (Fre.) Leung, T. K. (Department de Pathologie, Universite de Montreal, C.P. 6128, Montreal 101, Quebec, Canada); Bapai, F. *J Microsc Biol Cell* 23(2): 58a; 1975.

2916 STRUCTURE AND ULTRASTRUCTURE OF PROLACTIN ADENOMAS OF THE HUMAN HYPOPHYSIS (Abstract). (Fre.) Racadot, J. (Faculte de Medecine Pitie-Salpetriere, 105, boulevard de l'Hopital, 75634 Paris Cedex 13, France); Vila-Porcile, E.; Olivier, L.; Peillon, F. *J Microscop Biol Cellul* 23(2): 70a-71a; 1975.

2917 OSTEOCHONDROMA FOLLOWING IRRADIATION. CASE REPORT AND REVIEW OF THE LITERATURE. (Eng.) Pogrund, H. (Hebrew Univ.-Hadassah Medical Sch., Jerusalem, Israel); Yosipovitch, Z. *Israel J Med Sci* 12(2): 154-157; 1976.

2918 PIGMENTED PILOMATRICOMA (Abstract). (Fre.) Bourlond, A. (Louvain, Belgium). *Bull Soc Fr Dermatol Syphiligr* 82(3): 271; 1975.

2919 CLINICAL AND STATISTICAL STUDY OF 100 CASES OF VITILIGO. II. ASSOCIATED LESIONS. (Fre.) Ortonne, J.-P. (Hopital Edouard-Herriot, 69374 Lyon Cedex, France); Perrot, H.; Thivolet, J. *Sem Hop Paris* 52(11): 679-686; 1976.

2920 IMMUNOLOGIC ABNORMALITIES IN A PATIENT WITH MULTIPLE NEOPLASMS. (Eng.) Ko, H.-S. (Wellesley Hosp., Toronto, Ontario, Canada); Minkarious, E. F. *Can Med Assoc J* 114(10): 917-919; 1976.

See also:

* (Rev): 2416, 2429, 2436, 2443, 2444, 2445, 2446, 2447, 2448, 2449, 2452, 2453, 2455, 2456, 2457, 2459, 2460, 2461, 2462, 2463, 2469, 2471, 2491, 2498, 2501, 2502, 2503, 2504, 2505, 2506, 2507, 2510
* (Chem): 2521, 2540, 2542, 2548, 2549, 2550, 2561, 2595
* (Phys): 2624, 2629
* (Viral): 2640, 2643, 2657, 2661, 2662, 2702, 2710, 2712, 2716, 2719, 2728, 2735, 2736
* (Immun): 2757, 2767, 2782, 2783, 2813, 2814
* (Epid-Biom): 2924, 2928, 2930, 2933, 2935, 2940, 2946, 2947, 2950, 2952, 2953, 2954

2921 ASSOCIATION OF CADMIUM WITH RENAL CANCER. (Eng.) Kolonel, L. N. (Cancer Center Hawaii, Univ. Hawaii, Biomedical Sciences D-102, Honolulu, HI 96822). *Cancer* 37(4): 1782-1787; 1976.

Sixty-four cases of renal cancer in white males were compared with controls (197 cases of nonmalignant diseases of the digestive system and 72 cases of colon cancer) for past exposures to cadmium. Controls were also white males, and were group-matched to the cases on age for the analyses. Data on the three main sources of exposure to cadmium--diet, cigarette smoking, and occupation--were obtained by interview. The results showed a significant association of renal cancer with exposure to cadmium, and favored a synergistic effect between occupational exposure and smoking. The relative risk for men who both worked in high-risk occupations and smoked was more than four times that for men who did neither. The apparent synergism, however, suggests that the agent in cigarette smoke that contributes to this association may not be cadmium.

2922 A RETROSPECTIVE STUDY OF RENAL CANCER WITH SPECIAL REFERENCE TO COFFEE AND ANIMAL PROTEIN CONSUMPTION. (Eng.) Armstrong, B. (Radcliffe Infirmary, Oxford OX2 6HE, Great Britain); Garrod, A.; Doll, R. *Br J Cancer* 33(2): 127-136; 1976.

An interview of 106 patients with adenocarcinoma of the renal parenchyma, 33 patients with carcinoma of the renal pelvis, and 139 individually matched control patients was conducted. Comparison of the cancer patients with the control patients showed no evidence of a positive association between either type of renal cancer and coffee or animal protein consumption. However, a statistically significant negative association between cheese consumption and cancer of the renal pelvis suggested that animal protein may protect against this type of cancer by increasing the metabolic rate of carcinogens. Carcinoma of the renal pelvis was associated positively with cigarette consumption (relative risk estimate 1.8), and the daily consumption of analgesic tablets was more frequent in patients with cancer of the renal parenchyma than in their matched controls (14.2% compared with 1.9%). It appeared likely that the latter relationship was noncausal.

2923 TYPES OF LEUKEMIA IN CHRONIC BENZENE POISONING. A STUDY IN THIRTY-FOUR PATIENTS. (Eng.) Aksoy, M. (Internal Clinic of Istanbul Medical Sch., Capa, Istanbul, Turkey); Erdem, S.; Dincol, G. *Acta Haematol (Basel)* 55(2): 65-72; 1976.

The types of leukemia occurring in 34 individuals chronically exposed to benzene were studied and compared with the types of leukemia found in 50 nonexposed leukemic patients. Acute myeloblastic leukemia was the most frequent variety of this malignancy found in the subjects exposed to benzene; this was followed by preleukemia, erythroleukemia, and acute lymphoblastic leukemia. A preceding pancytopenic period was observed in 23.5% of all benzene-exposed cases, being conspicuously present in 50% of the preleukemic patients. The interval between the onset of the pancytopenic period and the onset of leukemia varied from 6 mo to 6 yr. Among 28,500 shoe-workers chronically exposed to benzene, the incidence of leukemia over an 8 yr period was 13.5/100,000; the comparable incidence in the general population was 6/100,000. In 3 of the 34 leukemic patients exposed to benzene, a genetic predisposition was established. In 6 of 8 new patients with acute leukemia, corticosteroids were used during the course of the pancytopenic period, and re-exposure to benzene was noted in four of the six patients. Among persons exposed to ionizing radiation, chronic lymphoid leukemia and chronic myeloid leukemia have been reported to occur frequently; the rarity of these forms of leukemia among persons exposed to benzene is therefore significant.

2924 ENHANCEMENT OF LYMPHOSARCOMA TRANSMISSION IN *ESOX LUCIUS* AND ITS EPIDEMIOLOGIC RELATIONSHIP TO POLLUTION. (Eng.) Brown, E. R. (Chicago Medical Sch., Chicago, IL 60612); Dolowy, W. C.; Sinclair, T.; Keith, L.; Greenberg, S.; Hazdra, J. J.; Beamer, P.; Callaghan, O. *Bibl Haematol (Basel)* (43): 245-251; 1976.

The relationship between malignant tumors in fish and the degree of pollution of the waterways in which they live was compared in three waterways of greatly different pollution. The correlation was significant ($p < 0.01$). Northern pike (*Esox lucius*) developed tumors after they were inoculated with Mulcahy reticulum cell sarcoma. Apparently cell-free extracts also cause tumor production, supporting the hypothesis of the viral etiology of this tumor, although Millipore-filtered material failed to produce tumors in young fish. The speed of tumor development is affected by inoculum size and site, water temperature, and age of recipient fish. Poliovirus was isolated from the gut of several fish from polluted water. (8 references)

2925 N-NITROSODIMETHYLAMINE IN AIR. (Eng.) Fine, D. H. (Thermo Electron Corporation, 85 First Ave., Waltham, MA 02154); Rounbehler, D. P.; Pellizzari, E. D.; Bunch, J. E.; Berkley, R. W.; McCrae, J.; Bursey, J. T.; Sawicki, E.; Krost, K.; DeMarrais, G. A. *Bull Environ Contam Toxicol* 15(6): 739-746; 1976.

The environmental distribution of N-nitroso compounds has been placed in a new perspective since N-nitrosodimethylamine (DMN) was found in the air in two U.S. cities, Baltimore, MD, and Belle, WV. Using two entirely different collection and analysis techniques, further extensive experiments in the two cities are reported. One analytical team collected samples cryogenically and analyzed the samples on a thermal energy analyzer. The second team concentrated organic vapors on preconditioned Tenax gas chromatography (35/60-mesh) cartridges. In Baltimore, much of the sampling was done near the Food Manufacturing

Corporation, which manufactures unsymmetrical dimethylhydrazine, for which DMN is an intermediate. A high value (32,000-36,000 ng/m^3) of DMN was found on the adjacent property. Samples collected in Arundel County revealed a decreasing DMN level in early morning. In downtown Baltimore, the opposite trend was observed. In Belle, where both DuPont and Union Carbide manufacture DMN, the levels found were lower than those found in Baltimore. Wastewater from DuPont and Fike Chemical Co. were found to contain DMN at concentrations of 2,000-4,000 ng/l. (16 references)

2926 ALIPHATIC HYDROCARBONS AND NAPHTHALENES AS WELL AS 3,4-BENZOPYRENE IN MUSSELS REMOVED FROM A COASTAL ZONE POLLUTED BY AN OIL SPILL. (Fre.) Bories, G. (Institut national de la Recherche agronomique, Laboratoire de Recherches sur les Additifs alimentaires, 180, chemin de Tournefeuille, 31300 Toulouse, France); Tulliez, J.; Peltier, J.-C.; Fleckinger, R. *C R Acad Sci [D] Paris* 282(17): 1641-1644; 1976.

Following an oil pollution of a coastal zone, the native mussels (*Mytilus edulis*) were studied for contamination with aliphatic hydrocarbons, naphthalenes, and 3,4-benzopyrene (BAP). Mussels were gathered 9, 35, 42, and 54 days after the pollution. Commercial mussels from another area were analyzed for comparison. Hydrocarbons were extracted from the saponified mussels, purified on a silica column, and measured by gas chromatography. BAP was measured by spectrofluorimetry using a portion of the saponified mussels. A sample of the oil responsible for the contamination was also analyzed. Contamination with hydrocarbons and naphthalenes on the ninth day was 0.1% of mussel weight. Contamination with BAP was on the order of 50×10^{-6}. BAP was eliminated from the mussels more rapidly than were the hydrocarbons and naphthalenes, which were eliminated between the 42nd and 54th day. Since mussels are a source of food, contamination by potential carcinogens is highly undesirable. (5 references)

2927 THE NEW ORLEANS DRINKING WATER CONTROVERSY. A STATISTICAL PERSPECTIVE. (Eng.) DuRouen, T. A. (Dept. Biostatistics, Univ. Washington, Seattle, WA 98195); Diem, J. E. *Am J Public Health* 65(10): 1060-1062; 1975.

The report released by the Environmental Defense Fund in November 1974 stating that ''over 50 premature deaths from cancer among white males would be averted annually'' by purifying or changing the source of the New Orleans water supply is examined. The figures of the report were arrived at by multiple regression analysis. In subsequent analyses by the same group, the water variable differed significantly from zero in regression analyses performed on total cancer for nonwhite men and women and white men, but not for white women. In two analyses in which significance was achieved for the water variable, the coefficient of determination for the entire regression equation was less than 0.15, indicating that the equation accounted for less than 15% of the variability in the cancer mortality rates. Thus the evidence supporting the hypothesis of water as the causative agent shows considerable inconsistency. In addition, the original report indicated that 9 of the 45 US counties having the highest cancer mortality rate were in Louisiana, but overlooked in the regression analysis that 4 of them do not use the Mississippi as a water source. Another report has used the same data to support a different hypothesis, citing ethnic and geographic differences as causes of variations in cancer mortality in the area. It is concluded that although the hypothesis put forth by the report is plausible, the evidence supporting it is not as strong as the publicity about it might suggest.

2928 CHROMOSOMAL EFFECTS IN PERIPHERAL LYMPHOCYTES. (Eng.) Purchase, I. F. H. (ICI Central Toxicology Lab., Alderley Park, near Macclesfield, Cheshire, England); Richardson, C.; Anderson, D. *Proc R Soc Med* 69(4): 290-292; 1976.

Eighty workers, including those exposed to polyvinyl chloride, nonexposed workers from the same site, and unexposed controls, were examined for chromosomal abnormalities in their peripheral lymphocytes. Results reveal an increase in abnormalities and a higher percentage of B and C cells in exposed workers than in the controls. Further studies with mice indicated that, even at lethal doses, there were no mutagenic effects in the germ cells.

2929 LEUKEMIA AND LYMPHOMA PATIENTS LINKED BY PRIOR SOCIAL CONTACT. EVALUATION USING A CASE-CONTROL APPROACH. (Eng.) Schimpff, S. C. (Natl. Cancer Inst., Univ. Maryland Hosp., Baltimore, MD); Brager, D. M.; Schimpff C. R.; Comstock, G. W.; Wiernik, P. H. *Ann Intern Med* 84(5): 547-550; 1976.

The social linkages between leukemia or lymphoma patients and a set of matched controls in each of two areas with low population mobility were studied. In the first area, for each of eight patients, a control matched for age, sex, race, and geographic location was chosen. In the second area, 21 patients were matched with controls. Close personal associations could include direct household contacts, social friendships, or other close social interactions (e.g., working together at a business establishment). Direct associations were defined as associations between two patients (or controls) or between a patient and a control. Indirect associations were defined as those that had one intermediary who had close contacts with both individuals for the same time period. Effective contact was defined as occurring before the onset of disease in one member of each pair of socially linked patients or controls. In the first area the patients had three direct and eight indirect personal links; the controls had no direct and two indirect links; and there were no links between patients and controls. The direct links were not significantly more than expected, but the total of 11 direct and indirect links was significantly more than the 3.25 expected. Two of the patients were blood relatives; none of the others were related by blood or marriage. In the second area there were 15 direct and 6 indirect links between patients and 8 direct and 3 indirect links between controls. There was a total of 28 links between cases and controls. The 21 links between patients was not significantly greater than the 14.75 expected. None of the patients were blood relatives, but two of the controls were. The findings, in part, lend support to previous observations that patients with leukemia or lymphoma are frequently interlinked by close personal associations.

2930 BURKITT'S DISEASE ON THE IVORY COAST: ANATOMO-CLINICAL AND EPIDEMIOLOGICAL CONSIDERATIONS. (Fre.) Loubiere, R. (B. P. 20632, Abidjan, Cote-d'Ivoire); Ette, M.; Vilasco, J.; Bonhomme, J. S.; Bureau, J. P. *Ann Anat Pathol (Paris)* 20(5): 437-452; 1975.

The epidemiology of Burkitt's lymphoma in the Ivory Coast is discussed from the aspect of climate. In the 4-yr period 1966-1970, Burkitt's lymphoma represented 4.12% (49/1,188) of cancers registered at Abidjan and 29% of cancers of children under the age of 15 yr. Despite the limitation of its occurrence in children only, it was the 6th most frequent neoplasm in the Ivory Coast. The maximum number of cases occurred at ages 6-8, but the tumor was observed in children under 1 yr and, exceptionally, at age 17. It was more likely to occur in boys by a factor of 2.6. The most frequent anatomical site was the face (80%). Any visceral organ can be a target for the lymphoma, but the kidney, adrenal gland, digestive tract, ovary, or CNS were the more common sites. Geographically, the Ivory Coast can be divided into equatorial forest areas and grasslands, and Burkitt's lymphoma was more prevalent in the forest districts (1/30,300 compared to 1/165,800). Of several climatic parameters studied for each district, only higher relative humidity correlated positively with frequency of cases. A parasite vector, such as malaria or trypanosomiasis, for the viral-induced Burkitt's lymphoma is considered probable. (18 references)

2931 CANCER IN UNITED STATES JEWS. (Eng.) Greenwald, P. (Bureau Cancer Control, New York State Dept. Health, Albany, NY 12237); Korns, R. F.; Nasca, P. C.; Wolfgang, P. E. *Cancer Res* 35(11/Part 2): 3507-3512; 1975.

Standardized mortality ratios for cancer of selected sites for Russian-born residents of upstate New York (1969 through 1971) were presented as an indirect measure of the problem in the United States Jews. Statistically significant excesses were found in men for stomach and colon, with a striking deficit in cancer of the buccal cavity and pharynx. Among women, excesses were noted for stomach, pancreas, and lung with a sharp deficit in the uterine cervix. On the basis of the religious affiliation of the cemetery of burial, estimates of the Jewish and non-Jewish components of the 800 deaths in Russian-born residents were determined. Expected deaths in these two subgroups by sex, for each cancer site, were then calculated by use of the site-specific proportionate mortality of upstate New York for these years. This revealed a significant excess among Jewish men for colon cancer, with a deficit in lung cancer; among the non-Jewish male components, stomach cancer mortality was the only site significantly in excess. Among Jewish women, stomach and lung cancers were in excess, with a deficit in cancers of the breast and cervix uteri. In non-Jewish Russian-born women, the only site significantly in excess was the stomach (with breast cancer showing a deficit).

2932 HEPATOCELLULAR CARCINOMA IN THE MALAYSIAN ORANG ASLI. (Eng.) Sumithran, E. (Faculty Medicine, Univ. Malaya, Kuala Lumpur, Malaysia); Prathap, K. *Cancer* 37(5): 2263-2266; 1976.

Necropsies were performed on 285 consecutively unclaimed Orang Asli bodies during an 8-yr period from May 1967 to April 1975. Of the 25 malignant neoplasms, hepatocellular carcinoma was the most common (nine patients, 36%). These patients had concurrent macronodular cirrhosis. There were 20 cases of cirrhosis; 45% of these had coexistent hepatocellular carcinoma. Of the 53,000 Orang Aslis living in West Malaysia comprising three tribes, the Negrito, Senoi, and Melayu Asli (Proto Malays), the Senoi appear to have a high predilection for liver cancer (all nine cases were Senoi) and cirrhosis (19/44 males), especially in males. These aboriginal people live in the jungles, where they practice shifting cultivation and maintain their own dietary and social customs. Detailed studies of their dietary habits may provide a clue to the etiology of liver cancer in these people.

2933 THE ASSOCIATION BETWEEN COLORECTAL CANCER AND BREAST CANCER. (Eng.) Howell, M. A. (Div. Cancer Cause and Prevention, Natl. Cancer Inst., Bethesda, MD 20014). *J Chronic Dis* 29(4): 243-261; 1976.

Evidence for an association between colorectal cancer and breast cancer is reviewed. Consistent with a dietary hypothesis to account for a relationship between the cancer sites, intestinal and breast cancers show a similar pattern in correlations of cancer mortality and incidence rates with *per capita* food consumption figures. Meat, particularly cattle meat, is the food most closely associated with colorectal and breast cancer rates. In the one case-control study that has shown dietary factors to be related to colorectal cancer, the specificity of beef as the most suspect food item implied the need for studies of long-chain, saturated fatty acids. A high animal fat and protein diet could also mean elevated levels of dietary tryptophan, the metabolism of which may be the biochemical mechanism for the interaction of nutritional and hormonal factors. A hormonal mechanism affected by dietary fat has similarly been postulated and has some experimental support. Possible research to further explore an association between colorectal and breast cancers is suggested.

2934 PLASMA HORMONE PROFILES IN POPULATIONS AT DIFFERENT RISK FOR BREAST CANCER. (Eng.)

Hill, P. (American Health Foundation, Valhalla, NY 10595); Wynder, E. L.; Helman, P.; Hickman, R.; Rona, G. *Cancer Res* 36(6): 1883-1885; 1976.

To determine whether the development of breast cancer in low- and high-risk populations is associated with changes in hormone profiles and also whether it is related to preexisting hormone levels in women at low risk for breast cancer were compared with gonadotropin levels in young premenopausal Bantu women at low risk for breast cancer were compared to the levels in high-risk Caucasian women. The subjects were healthy women (83 Bantu, 20 Caucasian) and patients with advanced breast cancer (11 Bantu, 12 Caucasian) up to 30 yr old. The plasma estradiol level was significantly lower in Bantu, but not in Caucasian, women with breast cancer, while the proportion of plasma androstenedione to dehydroepiandrosterone increased in Bantu patients. These changes in the plasma hormone profile in Bantu patients imply a change in ovarian and/or adrenal activity. It remains to be determined whether a change in ovarian function, in adrenal metabolism, or in ovarian dysfunction, together with altered adrenal metabolism, is associated with the development of breast cancer. Lack of similar hormone changes in Caucasian patients may be due to the initial differences in the hormone profile between healthy Caucasian and Bantu women and the different environmental factors that promote the breast disease in Western women.

2935 FAMILIAL BREAST CANCER. (Eng.)

Langlands, A. O. (Edinburgh Royal Infirmary, Edinburgh, Scotland); Kerr, G. R.; Bloomer, S. M. *Clin Oncol* 2(1): 41-45; 1976.

A study was made of 165 breast cancer patients who had one or more first degree relatives with the disease. The patients were compared with 2833 control cases. There was an improved survival experience in the familial group largely due to an earlier age of onset, an increased proportion of premenopausal patients, and to the occurrence of the disease in the familial group at an earlier stage. Though the familial cases occurred an average of 3-5 yr earlier than controls, more marked age differences were seen when the cases were subdivided according to their family relationship. The disease occurred significantly earlier than in controls in the daughters of breast cancer probands. It occurred significantly later than in controls in the mothers of breast cancer probands.

2936 THE INCREASING INCIDENCE OF MELANOMA.

(Eng.) Cosman, B. (161 Fort Washington Ave., New York, NY 10032); Heddle, S. B.; Crikelair, G. F. *Plast Reconstr Surg* 57(1): 50-56; 1976.

The records of Columbia-Presbyterian Medical Center and the statistics of New York City, New York State, and Connecticut were examined, for verification of an apparent increase in the number of melanomas. Between 1947 and 1973, 223 men and 246 women were treated at the Presbyterian Hospital for melanoma. The number of cases seen per yr rose precipitously during this period, a trend which was confirmed by the New York State and Connecticut statistics. In New York, the rate increased 1.9 times for women and 2.6 times for men between 1950 and 1971, and in Connecticut, the rate rose 3.3-fold for men and 6.3-fold for women between 1935 and 1972. The Connecticut statistics also indicate that the mortality rate is rising slightly for both sexes, and that the poorer prognosis for the male melanoma patient has not changed. The number of melanoma deaths has also risen in New York City, despite a decreasing proportion of Caucasians in the city population. The locations of the lesions treated have not changed. Thus, it is unlikely that the more recently diagnosed lesions are biologically less significant than those diagnosed in the past or that the incidence of melanoma has been altered by some specific contact carcinogen. The increasing incidence of melanoma appears to be a real phenomenon, although the cause of the increase remains unknown.

2937 ESTROGENS AND ENDOMETRIAL CANCER IN A RETIREMENT COMMUNITY. (Eng.)

Mack, T. M. (Cancer Surveillance Programs, Edmondson Research Bldg., 1840 North Soto St., Los Angeles, CA 90032); Pike, M. C.; Henderson, B. E.; Pfeffer, R. I.; Gerkins, V. R.; Authur, M.; Brown, S. E. *N Engl J Med* 294(23): 1262-1267; 1976.

To try to confirm reports that conjugated-estrogen use is causally linked with endometrial carcinoma, various measures of drug use and the invasiveness of endometrial cancer were studied in cases and controls chosen from an entire population in an affluent retirement community. The risk ratio for any estrogen use was estimated from all available evidence to be 8.0 (95% confidence interval, 3.5 to 18.1), and that for conjugated estrogen use to be 5.6 (95% confidence interval, 2.8 to 11.1). Increased risk from estrogens was shown for invasive as well as noninvasive cancer, and a dose-response effect was demonstrated. For an estrogen user, the risk from endometrial cancer appeared to exceed by far the baseline risk from any other single cancer. Although no estrogens are completely safe, it is recommended that, where indicated, they be administered cyclically and in the smallest possible dose. Because of a latency of up to 8 yr and because of a recent upsurge in prescriptions, the increase in endometrial cancer can be expected to continue past 1980.

2938 SKIN CANCER IN SUNNY QUEENSLAND. (Eng.)

Harris, R. J. (Royal Brisbane Hosp., Queensland, Australia). *Br J Plast Surg* 29(1): 61-67; 1976.

The characteristics of skin cancer as seen in Queensland, Australia, which has the highest recorded incidence of skin cancer in the world, are summarized. These observations are based on the excision of

6,615 lesions believed to be skin cancer, from 2,420 patients over 7 yr. Only 47% of cases presented with a single skin cancer; 42% had 2-5; 8.45% had 11-20, and 0.1% (12 patients) had 21-50 lesions excised at a single operation. The total removed from any individual over a period may be many times greater. More lesions occurred after 40 yr of age but there were still many in the under-40 age group; the sex distribution was 70% male, 30% female. The distribution observed was: basal cell carcinoma, 58%; squamous cell carcinoma, 23%; keratoacanthomas, 0.7%; basisquamous carcinomas, 1.3%; hyperkeratoses, 12%; malignant melanomas, 0.7%; miscellaneous, 1.9%; and unknown histology, 2.4%. Most of the lesions in this series (78%) occurred on the head and neck. Basal cell carcinomas were treated by dermatological applications, radiotherapy, or excision, depending on the indications. There are insufficient surgeons and facilities to permit excision of all basal cell carcinomas presented in Queensland. New cases of squamous cell carcinomas were preferably excised, as were malignant melanomas. The cause is almost certainly exposure to subtropical and tropical sunlight of the fair skins of the inhabitants. Daily exposure during routine occupations is probably more significant than sunbathing. The appearance of skin lesions on teenagers indicates that prophylactic screening should begin in infancy and much more care should be taken during school days.

2939 REVERSIBLE ARREST OF CHINESE HAMSTER V79 CELLS IN G2 BY DIBUTYRYL CYCLIC AMP. (Eng.) Stambrook, P. J. (Dept. Biology, Case Western Reserve Univ., Cleveland, OH 44106); Velez, C. *Exp Cell Res* 99(1): 57-62; 1976.

Chinese hamster V79 cells cultured in the presence of N^6,O^2-dibutyryl cyclic AMP (db-cAMP) grew at a slower rate and to a lower saturation density than untreated control cells. After 48 hr of culture with the drug there was no increase in cell number. The cells remained viable, however, since upon removal of the drug the cell number increased to that of untreated control cultures. Within 1 hr after removal of the db-cAMP, up to 20% of the population was in mitosis. The mitotic index remained elevated for a total of 2-3 hr after which it fell 4-fold, suggesting that a population of cells had accumulated shortly before the onset of mitosis, either at the end of the S phase or in G2. Autoradiographic analysis of cells that were pulse-labeled with $[^3]$TdR at the time the drug was removed showed that none of the mitotic cells during the initial burst of mitosis was labeled, indicating that in the presence of db-cAMP 30-40% of these cells had accumulated in G2. About 30% of the cells apparently were in the S phase at the time of drug removal.

2940 CANCER RESISTANCE. (Eng.) Lynch, H. T.; Guirgis, H. In: *Cancer Genetics*. Edited by H. T. Lynch. (Springfield, IL: Charles C. Thomas): pp. 471-490; 1976.

Two families (A and B) in the general population that showed a profound paucity of cancer and no other known associated genetic trait or disorder are identified. Review of the literature concerning certain genetic disorders such as diabetes mellitus, osteogenesis imperfecta, and glucose-6-phosphate dehydrogenase deficiency revealed a negative correlation with cancer. A study of 4,000 consecutively ascertained normal subjects (5,000 families) demonstrated that a sizeable proportion of the population (approximately 46%) show an absence of cancer among first degree relatives, while a minority of the population (approximately 7%) are cancer prone, as evidenced by three or more occurrences of cancer in first degree relatives. These findings have implications for research in carcinogenesis, in that it may be as important to know why certain members of the population are at reduced risk for cancer as it is to know why those in the population are remarkably cancer prone. Cancer control implications also exist in that an important risk factor to be considered in any cancer control program is family history. This even applies to other obvious risk factors such as cigarette smoking. Finally, these studies indicate a wide range of cancer susceptibility in humans, a phenomenon that is a function of both genetic and nongenetic factors, and which is very similar to findings observed at the infrahuman level.

2941 ONCOLOGY. (Eng.) Tiffany, R. (Royal Marsden Hosp., London, England). *Nurs Mirror* 142(22): i-iv; 1976.

2942 OCCUPATIONAL DISEASES IN CONNECTION WITH THE MANUFACTURE OF PLASTIC BOTTLES FROM POLYVINYL CHLORIDE. (Fre.) Duport, J. (Centre de Recherches de l'I.N.R.S., B.P. 27, 54500 Vandoeuvre-les-Nancy, France); Andlauer, P.; Gattelet, M.; Chalabreysse, J.; Archimbaud, M.; Teulon, F.; Bertrand, R. *Arch Mal Prof Med Trav Secur Soc* 36(4-5): 225-241; 1975.

2943 ESTIMATING HEALTH RISKS IN STUDIES OF THE HEALTH EFFECTS OF ASBESTOS. (Eng.) Enterline, P. E. (Dept. Biostatistics, Univ. Pittsburgh, Pittsburgh, PA). *Am Rev Respir Dis* 113(2): 175-180; 1976.

2944 AN EPIDEMIOLOGICAL STUDY OF 450 PATIENTS WITH WARTS TREATED AT THE SMITH COLLEGE HEALTH SERVICE BETWEEN 1964 AND 1972. (Eng.) Edelberg, H. (Smith Coll., Northhapton, MA). *J Am Coll Health Assoc* 23(4): 267-271; 1975.

2945 EPIDEMIOLOGY OF HODGKIN'S DISEASE. (Eng.) Schottenfeld, D. In: *Hodgkin's Disease*. Edited by M. J. Lacher. (New York: John Wiley & Sons): pp. 5-29; 1976.

2946 CERVICAL CANCER SCREENING IN A JOHANNESBURG FAMILY PLANNING CENTRE. (Eng.) Leiman, G. (Family Planning Assoc. South Africa [Transvaal Region], Johannesburg, South Africa). *S Afr Med J* 50(16): 611-615; 1976.

2947 SQUAMOUS CARCINOMA OF THE LIP IN QUEENSLAND: A RELATIVELY BENIGN LESION. (Eng.) Harris, T. J. (Royal Brisbane Hosp., Queensland, Australia). *Br J Plast Surg* 29(1): 68-69; 1976.

2948 ORAL CANCER IN 57,518 INDUSTRIAL WORKERS OF GUJARAT, INDIA: A PREVALENCE AND FOLLOW-UP STUDY. (Eng.) Malaowalla, A. M. (Government Dental Coll. and Hosp., Ahmedabad, India); Silverman, S., Jr.; Mani, N. J.; Bilimoria, K. F.; Smith, L. W. *Cancer* 37(4): 1882-1886; 1976.

2949 FEMALE LUNG CANCER INCIDENCE IN A UNIVERSITY HOSPITAL. (Eng.) Murakami, M. (Sch. Medicine, Keio Univ., Tokyo, Japan). *Keio J Med* 24(2): 103-114; 1975.

2950 INVESTIGATIONS FOR SKIN ANTIBODIES IN MALIGNANT NEOPLASMS. (Eng.) Diederichsen, H. (Odense Univ. Hosp., Odense, Denmark); From, E.; Heydenreich, G. *Dermatologica* 151(2): 96-99; 1975.

2951 DYSPLASIA AND CARCINOMA IN SITU OF THE UTERINE CERVIX: PREVALENCE IN VERY YOUNG WOMEN (UNDER AGE 22). A ONE-YEAR STUDY IN A HEALTH PLAN POPULATION. (Eng.) Snyder, R. N. (Southern California Permanente Medical Group, 1510 N. Edgemont St., Los Angeles, CA 90027); Ortiz, Y.; Willie, S.; Cove, J. K. J. *Am J Obstet Gynecol* 124(7): 751-756; 1976.

2952 THE INCIDENCE AND CLINICAL SIGNIFICANCE OF RECTAL POLYPS. (Eng.) Payne, R. A. (North Middlesex Hosp., London, England). *Ann R Coll Surg Engl* 58(3): 241-242; 1976.

2953 MULTIPLE PRIMARY MALIGNANT NEOPLASMS IN GYNAECOLOGY. (Eng.) Rajhvajn, B. (Medical Faculty, Univ. Zagreb, Yugoslavia); Kurjak, A.; Beazley, J. M. *Br J Clin Pract* 30(3): 60-61, 66; 1976.

2954 SMALL-CELL DYSPLASIA AND IN SITU CARCINOMA OF THE BREAST: INCIDENCE AND AGE DISTRIBUTION IN PATIENTS BIOPSIED OVER A 29-YEAR PERIOD. (Eng.) Toker, C. (Mount Sinai Sch. Medicine, City Univ. New York, NY 10029). *Mt Sinai J Med NY* 43(3): 226-231; 1976.

2955 THE CELL PROLIFERATION KINETICS OF THE EMT6/M/AC MOUSE TUMOUR AT FOUR VOLUMES DURING UNPERTURBED GROWTH IN VIVO. (Eng.) Watson, J. V. (Univ. and M.R.C. Clinical Oncology and Radiotherapeutics Unit, Medical Sch., Hills Road, Cambridge CB2 2QH, England). *Cell Tissue Kinet* 9(2): 147-156; 1976.

See also:

* (Rev): 2404, 2405, 2406, 2411, 2413, 2416, 2428, 2450, 2451, 2454, 2457, 2459, 2460, 2475, 2476, 2484, 2497, 2501, 2506, 2507, 2508, 2509, 2511
* (Chem): 2517, 2540, 2542, 2549, 2557, 2562, 2567
* (Phys): 2627, 2631, 2634
* (Viral): 2729
* (Path): 2824, 2832, 2836, 2838, 2853, 2872, 2878, 2887, 2903, 2909, 2912

2956 ON THE REGULATION OF CELL GROWTH IN CULTURE. (Eng.) Skehan, P. (University Center Colorado Medical Center, Denver, CO 80220). *Exp Cell Res* 97(1): 184-192; 1976.

The conventional theory of density-dependent growth inhibition was assessed in the rat C_6 glioma cell line. Stocks of cells were fed with growth medium and 5% fetal calf serum, and determinations were made of growth curves, specific growth rates, contact number, and percent confluency of the cultures. The C_6 glioma cells showed an initial period of low growth rate followed by a period of growth acceleration, a period of maximal growth rate, a period of sharply decelerating growth, and a period of slowly decelerating growth. The density at which growth inhibition occurred declined as seeding density was reduced, while the average population density at maximum growth rate increased with seeding density. Growth inhibition occurred an average of 1 day after maximum growth rate was attained, and the beginning of growth rate inhibition occurred at average densities of 15-280%. Growth inhibition also occurred at a relatively fixed time after plating, the time of confluency being proportional to seeding density. The density regulation of growth rate amplitude operated not only at the time of maximum growth rate, but during the accelerating and decelerating phases of growth as well. Furthermore, density regulated the amplitude of the growth rate at all times following phase I, but did not cause inhibition (phase IV). Other data showed that neither the timing of growth inhibition nor the amplitude of the growth rate was regulated by medium conditioning, and neither the amplitude regulation nor the timing of growth events was influenced by extracellular matrix. No obvious relationship between cell contact and the onset of growth inhibition was observed. Thus, the growth of C_6 cells displays a pattern that differs from the conventional contact and density-inhibited models proposed for other cell lines; inhibition in this line is caused by an endogenous program which specifies the time base of growth events and is not subject to density-dependent regulation.

2957 CALCITONIN IN BREAST AND LUNG CANCER. (Eng.) Hillyard, C. J. (Dept. Experimental Pathology, Chester Beatty Res. Inst., London SW3, England); Coombes, R. C.; Greenberg, P. B.; Galante, L. S.; MacIntyre, I. *Clin Endocrinol (Oxf)* 5(1): 1-8; 1976.

Calcitonin was measured in extracts of breast carcinoma and other malignancies. Tissue was obtained from 31 patients: 21 with carcinomas of various sites, and 10 with breast conditions. Eight of the patients had medullary carcinomas of the thyroid and one also had a pheochromocytoma. One patient had an adenoid cystic tracheal carcinoma and four had lung carcinomas. Plasma samples from 47 patients with medullary carcinoma of the thyroid showed calcitonin levels in all cases above 0.5 ng/ml. Tissue was available for only 8 of these 47 patients. Of 28 patients with lung carcinomas, 18 had elevated plasma calcitonin and tumors from 4 patients with detectable calcitonin were extracted. All of the patients except one were selected preoperatively, after having been found to have elevated levels of plasma immunoreactive calcitonin. In contrast, breast cancer patients were unselected. Immunoreactive calcitonin was detected in all but one extract of malignant tumors. However, it was not detected in extracts of benign breast lesions, normal lung, or normal breast tissue. Medullary carcinoma tissue contained much higher concentrations of immunoreactive calcitonin than did the other tumors. In cases where two antisera were used for the analysis, calcitonin was detected with both, but the concentration found with antiserum HGB 14 was consistently higher than that obtained with antiserum A. These findings indicate that the tumors contained a material very similar to human calcitonin. However, the fact that larger quantities of immunoreactive calcitonin were found with antiserum HGB 14 than with antiserum A suggests that the material in extracts of nonthyroid tumors may differ from synthetic human calcitonin. The practical clinical implications of this study are great. For the first time, a hormone has been shown to be regularly associated with, and probably produced by, breast cancer. Studies are being performed to assess the value of plasma immunoreactive calcitonin estimations in the detection of residual tumor following mastectomy.

2958 A COMBINED BIOCHEMICAL AND HISTOCHEMICAL STUDY ON STEROID METABOLISM IN MOUSE OVARY AND OVARIAN TUMOURS. (Eng.) Snoeijing, R. (Dept. Pathology, Univ. Groningen, Oostersingel 63, Groningen, The Netherlands); Hardonk, M. J. *Acta Histochem (Jena)* 14: 119-128; 1975.

Steroid metabolism was studied, with the use of mouse ovaries and radiation-induced ovarian tumors to determine whether the histochemical determination of hydroxysteroid dehydrogenases corresponds with the biochemical findings. The histochemical reactions were done on 10 μ cryostat sections. The biochemical experiments were performed with homogenates or with cell fractions. Histochemically, 17β-hydroxysteroid dehydrogenase was observed in certain corpora lutea of normal mouse ovaries and not in granulosa cell- or interstitial-cell tumors. Biochemically, 17β-hydroxysteroid dehydrogenase activity was found in control ovaries, as well as in granulosa cell and interstitial cell tumors. In corpora lutea the two methods correlated well. 20α-Hydroxysteroid dehydrogenase activity was found in corpora lutea and varied according to the stage of development. Histochemically, tumors did not show any reaction, but biochemically some enzyme activity could be determined in granulosa cell and interstitial cell tumors. Normal ovaries had to be used for the 20α-hydroxysteroid dehydrogenase reaction; the results obtained with the two techniques correlate well. Because 3β-hydroxysteroid dehydrogenase is present in many cell types of a normal ovary, and its activity varies greatly from one cell type to another, granulosa cell and interstitial cell tumors were used in a combined experiment. The histochemical and biochemical data correlated very well. With interstitial tumors as a model and androstenedione as substrate, anabolic and/or catabolic steroids were synthesized. There was no correlation between 3β-hydroxysteroid dehydrogenase activity and production of anabolic steroids. A correlation was found between NADP-dependent dehydrogenase activities, especially malate dehydrogenase activity, and the production of catabolic steroids in interstitial cell tumors.

2959 THE RADIOIMMUNOASSAY (RIA). (Eng.) Van der Gugten, A. A. (Netherlands Cancer Inst., Amsterdam, The Netherlands). *Int Union Cancer Tech Rep Ser* 20: 130-134; 1975.

A radioimmunoassay technique for determining prolactin concentrations in plasma and pituitaries is described. Prolactin is isolated from prolactin-producing pituitary tumors transplanted into rats (2-4 rats yield 100 mg prolactin). The tumor-bearing castrated male rats are treated with estrone to support tumor growth. The prolactin is chromatographed on DEAE-cellulose. An antiserum to rat prolactin is raised in the rabbit. The prolactin is labeled with ^{125}I by adding 1 mCi of $Na^{125}I$ and 25 μl of chloramine-T to 10 μl of the prolactin solution. After purification on Sephadex G-50 column, the prolactin is stored in 0.05 ml aliquots at -20 C until needed for the radioimmunoassay. Since the radioimmunoassay is based upon the competition between a labeled antigen and the unlabeled antigen for binding to a restricted number of antibodies, a method to separate bound from free labeled hormone is necessary; a commonly used technique is the double antibody method. The dilution of antiserum containing just enough antibodies to bind the labeled hormone nearly completely is used in the assay of competitive inhibition. The extent to which labeled prolactin and unlabeled prolactin compete for the limited number of binding sites on the antibody can be measured by incubation of a fixed amount of labeled antigen and the fixed dilution of antiserum with serial dilutions of unlabeled antigen. If the concentration of unlabeled antigen is plotted on a log scale and the percentage of radioactivity in the complex on a linear scale, a regression curve is obtained, that becomes linear over a certain range. Reference curves for standard preparations are prepared and the amount of prolactin present in the plasma or pituitary homogenates is expressed as nanograms of the standard preparation. This method can be adapted to determine several antigens (mammary tumor virus or carcinoembryonic) of interest in cancer research.

2960 INTRACELLULAR DISTRIBUTION OF THE PROTEIN KINASE ACTIVITY IN NORMAL AND NEOPLASTIC RAT THYROID TISSUE. (Fre.) Meldolesi, M. F. (Centro di Endocrinologia ed Oncologia sperimentale del C.N.R., Universita di Napoli, Naples, Italy); Macchia, V.; Laccetti, P. *C R Soc Biol (Paris)* 169(6): 1442-1448; 1975.

The intracellular enzyme activity of tissue from normal rat thyroid and the transplantable thyroid neoplasm of the strain 1-5G Wollman rat was studied. The thyroid neoplasm is rapidly growing and is not sensitive to thyroid-stimulating hormone. Thyroids or neoplasms were removed from the rats, minced, and homogenized in a buffered solution containing tris-HCl, 0.05 M, and saccharose, 0.25 M; they were then centrifuged at different speeds to obtain the subcellular fractions. Concentrations of protein kinase, adenylate cyclase, and cytochrome oxidase were calculated for the whole homogenate, the soluble fraction, and the nuclear, mitochondrial, and microsomal fractions. The adenylate cyclase and cytochrome oxidase activity of normal and neoplastic tissue were similar. Protein kinase activity was greater in the mitochondrial and microsomal fractions of the normal thyroid and in the soluble and nuclear fractions of the thyroid neoplasm. Cyclic AMP, 0.1 nanomole, or cyclic guanosine 5'-monophosphate, 1 mole, stimulated the protein kinase activity of both normal and neoplastic thyroid tissue, particularly when 100 μg of histone were added to the medium. The protein kinase activity of total homogenate of neoplastic thyroid tissue was three times greater than that of normal tissue in the presence of histone and more activated by cyclic AMP. The enzyme protein kinase in rat thyroid tumor shows significant differences in cell distribution and sensitivity to cyclic nucleotides compared to normal rat thyroid tissue, which has an enzymatic activity analogous to that of the pituitary gland. (20 references)

2961 URIDINE 5'-DIPHOSPHATE-GALACTOSE:GLYCOPROTEIN GALACTOSYLTRANSFERASE ACTIVITY IN THE OVARIAN CANCER PATIENT. (Eng.) Bhattacharya, M. (Dept. Gynecology, Roswell Memorial Inst., Buffalo, NY 14263); Chatterjee, S. K.; Barlow, J. J. *Cancer Res* 36(6): 2096-2101; 1976.

Uridine 5'-diphosphate galactose:glycoprotein galactosyltransferase activity was measured in homogenates of normal ovary and ovarian epithelial adenocarcinomas. The specific activity was 3 to 5 times higher in the tumoral ovaries than in normal ones. The glycoprotein fetuin, from which terminal sialic acid and penultimate galactose were removed (fetuin minus N-acetylneuraminic acid and galactose), acted as an excellent exogenous acceptor. Galactosyltransferase from normal ovary and ovarian tumor cells had similar properties. Both required Mn^{2+} and Triton X-100 and had broad pH optima between 5.5 and 7. Galactosyltransferase activity was also measured in serum samples from ovarian cancer patients and normal healthy individuals in the presence of fetuin minus N-acetylneuraminic acid and galactose as exogenous acceptor. The enzyme levels were significantly elevated in the sera of ovarian cancer patients compared to normal controls. The differences in the levels of this enzyme in the tissues and sera of normal individuals and ovarian cancer patients were not the result of different levels of the degrading enzymes such as uridine 5-diphosphate galactose pyrophosphatase or β-D-galactosidase. Extended serial determinations revealed that the level of galactosyltransferase activity correlated with tumor volume and with the clinical status of the patient; this suggests possible leakage of the tumor enzyme into the host sera. Serial determination of this enzyme level in ovarian cancer patients may reflect tumor progression or success of therapeutic approaches.

2962 PREFERENTIAL BIOSYNTHESIS OF RIBOSOMAL STRUCTURAL PROTEINS BY FREE AND LOOSELY BOUND POLYSOMES FROM REGENERATING RAT LIVER. (Eng.)

Nabeshima, Y.-I. (Niigata Univ. Sch. Medicine, Niigata, Japan); Tsurugi, K.; Ogata, K. *Biochim Biophys Acta* 414(1): 30-43; 1975.

Homologous cell-free systems were prepared using free, total bound, tightly bound or KCl-sensitive loosely bound (KCl-sensitive) polysomes from regenerating rat liver. [^{14}C]Leucine was incubated with one kind of polysomes and [^{3}H]leucine with another kind. The reaction mixtures were then combined, and ribosomal structural proteins were purified, using two-dimensional polyacrylamide gel electrophoresis. The ^{3}H to ^{14}C ratios of the purified fractions were estimated to compare the activities of the two kinds of polysomes for biosynthesis of ribosomal structural proteins. The activity of free polysomes for biosynthesis of ribosomal structural proteins was about 3.6 or 2.4 times higher than that of total bound polysomes in two experiments in which ^{14}C and ^{3}H labeling was reversed. The radioactivities incorporated by free polysomes into most of the proteins separated on two-dimensional gel were found to be definitely higher than those in the surrounding areas, which suggested that most of the ribosomal structural proteins were synthesized by free polysomes. The activity of free polysomes for biosynthesis of ribosomal structural proteins was approximately sevenfold higher than that of tightly bound polysomes (which were prepared by washing the microsomal membrane fraction with 0.5 M KCl). The radioactivities incorporated by tightly bound polysomes into the proteins separated on two-dimensional gel were only slightly higher than those in the surrounding areas, which indicated that these polysomes had very low synthetic activity. Most of the ribosomal structural proteins were preferentially synthesized by free and KCl-sensitive polysomes in regenerating rat liver.

2963 HIGHER TRANSPEPTIDATION ACTIVITY AND BROAD ACCEPTOR SPECIFICITY OF γ-GLUTAMYLTRANSFERASES OF TUMORS. (Eng.)

Tateishi, N. (Osaka Univ. Medical Sch., Osaka, Japan); Higashi, T.; Nomura, T.; Naruse, A.; Nakashima, K.; Shiozaki, H.; Sakamoto, Y. *Gann* 67(2): 215-222; 1976.

The γ-glutamyl cycle has been proposed as an amino acid transport system. The γ-glutamyltransferase (γ-glutamyltranspeptidase, γ-GTP) activity of rapidly growing tissue was studied to determine whether the enzyme showed higher activity only or whether it acquired novel properties more adequate for amino acid transport. γ-GTP activity in rat hepatomas induced by 3'-methyl-4-(dimethylamino)azobenzene was 120-fold higher than that of normal liver; high activity was also found in bovine hepatocellular carcinoma. γ-GTP's from these malignant tissues responded more and showed broader specificity to γ-glutamyl group acceptors than those from normal liver and kidney tissue. Three species of γ-GTP were isolated from bovine kidney by DEAE-cellulose chromatography, whereas only two species were isolated from bovine hepatocellular carcinoma. The carcinoma lacked the least acidic enzyme species. Appropriate γ-glutamyl group acceptors stimulated more-acidic enzyme species to a greater degree than less-acidic species in both tissues. The fractions separated from the hepatoma were stimulated much more than those of the kidney by a γ-glutamyl group acceptor. The enzymes from normal tissues responded similarly to a γ-glutamyl group acceptor irrespective of the difference in their activity. Thus, γ-GTP's of malignant tissues appear to be more versatile for amino acid transport, both qualitatively and quantitatively. In these properties the enzyme of mouse fetal liver that showed the highest activity in the last period of pregnancy resembled the enzymes of malignant rather than normal tissues. Hepatic γ-GTP activity is not parallel with cell proliferation rate during normal development. (33 references)

2964 NOTES ON CELL CYCLE ANALYSIS AND CELL SYNCHRONIZATION. (Eng.)

Rajewsky, M. F. (Max-Planck-Institut fuer Virusforschung, Tuebingen, W. Germany). *Int Union Cancer Tech Rep Ser* 20: 52-62; 1975.

Several techniques for cell cycle analysis and cell synchronization and the preparation of cells for pulse-cytophotometry are briefly discussed. The most powerful and frequently used method for cell cycle analysis is the fraction of labeled mitoses technique, which can be used to estimate the durations of the different cell cycle periods and the distribution of cell cycle times of the individual proliferating cells in a given population. The technique consists of (a) pulse-labeling the DNA synthesizing cells of an asynchronously proliferating cell population in vivo or in culture with ^{3}H- or ^{14}C-labeled thymidine, (b) collecting cell or tissue samples at different times after labeling, (c) preparing cell smears or microtome tissue sections, (d) autoradiography of these preparations, and (e) recording of the ratio of labeled mitotic cells to the total number of mitotic cells. The ^{3}H-, ^{14}C-thymidine double labeling technique is used to estimate the duration of the S-period and to measure the intermitotic time in slowly renewing tissues. The time for the halving of the autoradiographic grain count over ^{3}H-thymidine-labeled nuclei is also a measure of intermitotic time, but the precision of the technique is limited by methodological uncertainties in grain count determinations. Continuous (or repeated) labeling with ^{3}H-thymidine can give information on the duration of the S-period, on the turnover time, and on the duration of the cell cycle. Other cell cycle analysis techniques involve age-distribution of proliferating cells and distribution of cells in different cell cycle phases according to their relative DNA content. The latter method consists of ''conversion to a single cell suspension,'' fluorochrome staining of DNA, and measurement of the DNA content by pulse-cytophotometry. Two types of method are available for synchronization of cell populations (a) selection (e.g. by sedimentation) of cells in subcompartments of the cell cycle; and (b) temporary arrest of cells in a given phase of the cell cycle (with use of colcemid or certain Vinca alkaloids).

2965 CHARACTERISTICS OF MUCIN-TYPE SIALOGLYCOPEPTIDE PRODUCED BY B16 MOUSE MELANOMA CELLS. (Eng.) Bhavanandan, V. P. (Milton S. Hershey Medical Center, Pennsylvania State Univ., Hershey, PA 17033); Davidson, E. A. *Biochem Biophys Res Commun* 70(1): 139-145; 1976.

2966 FEEDBACK CONTROL OF CELL GROWTH AND THE 2'-DEOXYURIDINE TRANSPORT SYSTEM (Proceedings Abstract). (Eng.) Wigler, P. W. (Univ. Tennessee Memorial Res. Center, Knoxville, TN 37920); Schluter, D. N. *IRCS Med Sci (Suppl)* 3(10): 8; 1976.

2967 IN VITRO CULTURE TECHNIQUES FOR TUMOUR CELLS. (Eng.) Metcalf, D. (Walter and Eliza Eliza Hall Inst., Melbourne, Australia). *Int Union Cancer Tech Rep Ser* 20: 106-109; 1975.

2968 THE MAXIMAL SIZE OF AVASCULAR TUMORS (Proceedings Abstract). (Eng.) Blumenson, L. E. (Roswell Park Memorial Inst., Buffalo, NY 14263); Bross, I. D. J. *Proc Am Assoc Cancer Res* 17: 1; 1976.

2969 CHOLESTEROL AND FATTY ACID PROFILES IN PLASMA MEMBRANES FROM MORRIS HEPATOMAS OF VARYING DEGREES OF MALIGNANCY (Proceedings Abstract). (Eng.) Dnistrian, A. M. (Memorial Sloan-Kettering Cancer Center, New York, NY 10021); Barclay, M.; Archibald, F. M.; Terebus-Kekish, O.; Morris, H. P. *Proc Am Assoc Cancer Res* 17: 3; 1976.

2970 UREA SYNTHESIS IN NOVIKOFF AND MORRIS HEPATOMAS (Proceedings Abstract). (Eng.) Lawson, D. (Fels Res. Inst. and Dept. Biochemistry, Temple Univ. Sch. Medicine, Philadelphia, PA 19140); Paik, W. K.; Morris, H. P.; Weinhouse, S. *Proc Am Assoc Cancer Res* 17: 54; 1976.

2971 RESOLUTION AND RECONSTITUTION OF HEPATOMA DRUG METABOLISM SYSTEMS (Proceedings Abstract). (Eng.) Saine, S. E. (Univ. Texas Medical Sch., Houston, TX 77025); Dignam, J. D.; Strobel, H. W. *Fed Proc* 35(7): 1654; 1976.

2972 EXPERIMENTAL MENINGEAL CARCINOMATOSIS (CA): DEVELOPMENT AND CHEMOTHERAPY (Proceedings Abstract). (Eng.) Ushio, Y. (Sloan-Kettering Inst., New York, NY 10021); Chernik, N.; Posner, J.; Shapiro, W. *Proc Am Assoc Cancer Res* 17: 32; 1976.

2973 ENHANCED TUMORIGENICITY IN T-333 CELLS GROWN IN PLASTIC (Proceedings Abstract). (Eng.) Lee, W.-Y. (Johns Hopkins Univ., Baltimore, MD 21205); Bang, F. B. *Proc Am Assoc Cancer Res* 17: 196; 1976.

2974 HUMAN HEPATOBLASTOMA IN CELL CULTURE (Abstract). (Eng.) Helson, L. (Memorial Sloan-Kettering Cancer Center, New York, NY 10021); Helson, C. *In Vitro* 12(4): 327-328; 1976.

2975 INDUCTION OF TUMOR GROWTH BY INOCULATION OF A HUMAN BREAST CANCER CELL (MCF-7) INTO OVARY OR PITUITARY GRAFTED NUDE MICE (Proceedings Abstract). (Eng.) Russo, J. (Dept. Biology, Michigan Cancer Foundation, Detroit, MI 48201); Brennan, M. J.; Rich, M. A. *Proc Am Assoc Cancer Res* 17: 116; 1976.

2976 PHOSPHOLIPID METABOLISM IN EHRLICH ASCITES TUMOR CELLS: I. TURNOVER RATE OF PHOSPHATIDYLINOSITOL. (Eng.) Waku, K. (Medical Res. Inst., Tokyo Medical and Dental Univ., Bunkyo-ku, Tokyo 113, Japan); Nakazawa, Y.; Mori, W. *J Biochem (Tokyo)* 79(2): 407-411; 1976.

2977 COMPARATIVE CHARACTERIZATION OF HUMAN MELANOMA CELLS IN VITRO (Proceedings Abstract). (Eng.) Creasey, A. A. (Cell Culture Lab., Univ. California, Sch. Public Health, Naval Biomedical Res. Lab., Oakland, CA 94625); Fukuyama, K.; Epstein, W. *Proc Am Assoc Cancer Res* 17: 189; 1976.

2978 CULTURED LUNG ADENOMA CELLS FOR THE ANALYSIS OF SURFACTANT BIOSYNTHESIS (Proceedings Abstract). (Eng.) Stoner, G. D. (Univ. California-San Diego, La Jolla, CA.92093); Hallman, M. N. *Proc Am Assoc Cancer Res* 17: 91; 1976.

2979 MAINTENANCE OF CYTODIFFERENTIATION OF DISSOCIATED MAMMARY EPITHELIAL CELLS OF FLOATING COLLAGEN MEMBRANES (Abstract). (Eng.) Emerman, J. T. (Univ. California, Berkeley, CA 94720); Pitelka, D. R. *In Vitro* 12(4): 329; 1976.

2980 THE ROLE OF ADENOSINE 3',5'-MONOPHOSPHATE IN THE TRANSFORMATION OF CLOUDMAN MOUSE MELANOMA CELLS. (Eng.) Lincoln, T. M. (Vanderbilt Univ. Sch. Medicine, Nashville, TN 37232); Vaughan, G. L. *J Cell Physiol* 86(3): 543-552; 1975.

2981 ISOLATION OF EPITHELIAL CELLS FROM HAMSTER TRACHEAL RESPIRATORY EPITHELIUM (Abstract). (Eng.) Ezerman, E. B. (Univ. Vermont, Burlington, VT 05401); Adler, K. B.; Craighead, J. E. *In Vitro* 12(4): 319; 1976.

2982 THE INFLUENCE OF CULTURE REGIME ON NEOPLASTIC TRANSFORMATION IN VITRO (Proceedings Abstract). (Eng.) Taylor, W. G. (Lab. Biochemistry, NCI, Bethesda, MD 20014); Camalier, R. F.; Kerr, A. *Proc Am Assoc Cancer Res* 17: 68; 1976.

2983 AGGREGATE PROPERTIES OF HUMAN SARCOMA CELLS: CORRELATION WITH GROWTH IN SOFT AGAR AND TUMORIGENICITY (Proceedings Abstract). (Eng.) Steuer, A. F. (Biotech Res. Lab., Rockville, MD 20852); Rhim, J. S.; Ting, R. C. *Proc Am Assoc Cancer Res* 17: 34; 1976.

2984 EFFECT OF N^6,$O^{6'}$-DIBUTYRYL CYCLIC AMP TREATMENT OF LYMPHOMA CELLS ON THE TRANSLATION OF GLOBIN mRNA BY CELL-FREE EXTRACTS. (Eng.) Negreanu, J. (Weitzman Inst. Science, Rehovot, Israel); Daniel, V. *FEBS Lett* 64(2): 423-427; 1976.

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National Cancer Institute

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PREFACE

Carcinogenesis Abstracts is a publication of the National Cancer Institute. The journal serves as a vehicle through which current documentation of carcinogenesis research highlights are compiled, condensed, and disseminated on a regular basis. It represents an integral part of the Institute's program of fostering and supporting coordinated research into cancer etiology. Issues of *Carcinogenesis Abstracts* normally contain three-hundred abstracts and three-hundred citations (unaccompanied by corresponding abstracts). Abstracts and citations refer to the current scientific literature that describes the most significant carcinogenesis research carried on at the National Cancer Institute, other governmental agencies, and private institutions. *Carcinogenesis Abstracts* is intended to be a highly useful current awareness tool for scientists engaged in carcinogenesis research or related areas. The great number and diversity of publications relevant to carcinogenesis make imperative the availability of this service to investigators whose work requires that they keep abreast with current developments in the field.

Carcinogenesis Abstracts is normally published monthly. Volume XIV covers the scientific literature published from Jan 1976 through Dec 1976. To increase the usefulness of *Carcinogenesis Abstracts*, Volume XIV, a Wiswesser Line Notation index and a Chemical Abstracts Service Registry Number index have been provided. These indexes reference compounds described in abstracted articles. A cumulative subject, author, CAS Registry Number, and Wiswesser Line Notation index for Volume XIV will be published shortly after the final regular issue.

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NOTE

Journal names are abbreviated according to the list of abbreviations used by *Index Medicus*. For journals not covered by *Index Medicus*, the abbreviations found in *Chemical Abstracts Service Source Index*, 1907-1974 Cumulative, are used. New journals are verified in *New Serial Titles* and abbreviated according to *International Standard ISO 833*. An asterisk indicates the author to address (other than the primary) in requesting reprints.

LANGUAGE ABBREVIATIONS

Afr.	Afrikaans	Ind.	Indonesian
Ara.	Arabic	Ita.	Italian
Bul.	Bulgarian	Jpn.	Japanese
Chi.	Chinese	Kor.	Korean
Cro.	Croation	Lav.	Latvian
Cze.	Czech	Lit.	Lithuanian
Dan.	Danish	Nor.	Norwegian
Dut.	Dutch	Pol.	Polish
Eng.	English	Por.	Portuguese
Est.	Estonian	Rum.	Rumanian
Fin.	Finnish	Rus.	Russian
Fle.	Flemish	Ser.	Serbo-Croatian
Fre.	French	Slo.	Slovak
Geo.	Georgian	Spa.	Spanish
Ger.	German	Swe.	Swedish
Gre.	Greek	Tha.	Thai
Heb.	Hebrew	Tur.	Turkish
Hun.	Hungarian	Ukr.	Ukrainian
Ice.	Icelandic	Vie.	Vietnamese

ABBREVIATIONS USED IN ABSTRACTS

A	angstrom(s)	M	molar
ACTH	adrenocorticotropic hormone	mM	millimolar
ADP	adenosine diphosphate	μM	micromolar
AMP	adenosine monophosphate	mOsm	milliosmolar
ATP	adenosine triphosphate	mEq	milliequivalents
BCG	Bacillus Calmette Guerin	min	minute(s)
bid	twice daily	mo	month(s)
C	degrees centigrade	MTD	maximum tolerated dose
cal	calorie(s)	N	normal concentration
kcal	kilocalorie(s)	NAD	nicotinamide adenine dinucleotide
cc	cubic centimeter(s)	NADH	reduced nicotinamide adenine dinucleotide
Ci	curie(s)	NADP	nicotinamide adenine dinucleotidephosphate
mCi	millicurie(s)	NADPH	reduced nicotinamide adenine dinucleotidephosphate
μCi	microcurie(s)		
cm	centimeter(s)	ng	nanogram(s) (10^{-9})
CNS	central nervous system	od	once daily
cpm	counts per minute	Pa	ambient pressure
dl	deciliter(s)	PAS	periodic acid-Schiff
ml	milliliter(s)	pg	picogram(s) (10^{-12})
μl	microliter(s)	pgEq	picogram equivalent
DNA	deoxyribonucleic acid	po	orally
ED_{50}	median effective dose	ppb	parts per billion
EDTA	ethylenediamine tetraacetic acid	ppm	parts per million
ESR	erythrocyte sedimentation rate	qid	four times daily
g	gram(s)	qod	every other day
kg	kilogram(s)	QO_2	oxygen quotient
mg	milligram(s)	R	roentgen(s)
μg	microgram(s)	RBC	red blood cells (erythrocytes)
Hb	hemoglobin	RNA	ribonucleic acid
hr	hour(s)	sc	subcutaneous
ia	intra-arterial	sec	second(s)
ic	intracerebral	SGOT	serum glutamic-oxalacetic transaminase
icav	intracavitary	SGPT	serum glutamic-pyruvic transaminase
id	intradermal	SRBS	sheep red blood cells
ILS	increased life span	TCD	tissue culture dose
im	intramuscular	TCD_{50}	median tissue culture dose
ip	intraperitoneal	tid	three times daily
ipl	intrapleural	U	unit(s)
it	intratumorous	mU	milliunit(s)
IU	International Unit	UV	ultraviolet
iv	intravenous	vol	volume
K_m	Michaelis constant	WBC	white blood cells (leukocytes)
LD	lethal dose	wk	week(s)
LD_{50}	median lethal dose	wt	weight
m	meter(s)	x	times
mm	millimeter(s)	yr	year(s)

3001 COMPARATIVE MEDICINE: PROGRESS AND PROSPECTS. (Eng.) Bustad, L. K. (Washington State Univ. Coll. Veterinary Medicine, Pullman, WA 99163); Gorham, J. R.; Hegreberg, G. A.; Padgett, G. A. *J Am Vet Med Assoc* 169(1): 90-104; 1976.

Comparative medicine has provided many clues to the etiology of cancer. Research on chemical agents as carcinogens has been questionable because of the use of different test systems and species. An appropriate test system that includes the latent period for cancer induction is also lacking at present. Viral research has revealed more promising results. In several species, a type C virus with RNA as its genetic material was implicated in causing a leukemia-sarcoma complex. Virus-induced primate tumors in apes are currently of great interest, because a virus recently reported in a human lymphosarcoma resembles the primate tumor virus. Animal experiments have contributed much to the understanding of physical agents as carcinogens. Studies on radium intoxication in mice and dogs were comparable to data from humans who received accidental exposure. Data from animal studies have enabled limits to be set for radium exposure, although the mechanism of radium tumorigenesis is not yet fully defined. (197 references)

3002 ENVIRONMENTAL CANCER. (Eng.) Weisburger, J. H. (American Health Foundation, Valhalla, NY 10595). *J Occup Med* 18(4): 245-252; 1976.

The types, causes, and incidences of environmental cancer are reviewed, along with positive steps for prevention and detection. The data revealed that 80%-90% of human cancers are caused by man-made environmental factors. Carcinogens may be: (a) direct-acting, causing cancer themselves; (b) procarcinogens, requiring host-mediated biochemical activation; or (c) cocarcinogens, not cancer producing but able to potentiate the effects of other carcinogens. The major causes of human cancer are use of tobacco products, dietary habits, and occupational exposure. The most effective ways to prevent cancer caused by tobacco use and dietary habits are (a) to educate the public as to steps individuals should take, such as using low tar and nicotine tobacco products and eating less fat and cholesterol; and (b) to persuade manufacturers to produce less harmful products. Occupational cancer can be lowered by controlling exposure to harmful agents and selecting older (and therefore less sensitive) individuals for employment in situations where contact with carcinogens might occur. (16 references.)

3003 IMPORTANCE OF GENETIC FACTORS INFLUENCING THE METABOLISM OF FOREIGN COMPOUNDS. (Eng.) Nebert, D. W. (Natl. Inst. Child Health Human Development, Natl. Insts. Health, Bethesda, MD 20014); Felton, J. S. *Fed Proc Fed Am Soc Exp Biol* 35(5): 1133-1141; 1976.

Past and current literature on the genetic differences in the metabolism of xenobiotics between strains of laboratory animals is reviewed. Gene differences may alter an individual's response to foreign compounds by affecting their absorption, binding, distribution, excretion, biotransformation, or drug-drug interactions. The inbred mouse has been studied most extensively. Genetic differences in toxicity were caused by various environmental pollutants in several inbred strains of mice and in siblings of the (C57BL/6N)(DBA/2N)F_1 x DBA/2N backcross, in which the phenotypes' ''aromatic hydrocarbon responsiveness'' or ''nonresponsiveness'' had been predetermined. This trait of ''responsiveness'', which refers to the capacity for induction of cytochrome P-450 and numerous monooxygenase activities by certain aromatic compounds, segregates almost exclusively as a single gene among offspring of this backcross. All nonresponsive mice ingesting benzo[a]pyrene (about 125 mg/kg/day) die within 4 wk, whereas the survival of responsive mice receiving the chemical po is not significantly different from that of control mice; the apparent cause of early death in these experiments is toxic depression of the bone marrow. The lifespan of animals exposed to certain environmental pollutants can therefore be markedly influenced by a single gene or a very small number of genes. The same genetic trait can be either beneficial or detrimental to the animal, depending on whether detoxification or metabolic potentiation occurs. There may also be genetic differences in man's susceptibility to toxicity or cancer caused by the numerous foreign compounds in his environment. (151 references)

3004 THE IMPACT OF DRUG METABOLISM ON MEDICINAL RESEARCH. PART 1: SURVEY OF METABOLIC PROCESSES. (Eng.) Parke, D. V. (Dept. Biochemistry, Univ. Surrey, Guildford, UK). *Chem Ind* (9): 380-388; 1976.

The relationship of drug metabolism to carcinogenesis is discussed, and a technique for measuring the carcinogenic potential of a chemical is described. Enzyme induction could be a factor in carcinogenesis since it may facilitate the establishment of a clone of malignant cells from a single malignantly transformed cell resulting from arylation of DNA by polycyclic hydrocarbons and other carcinogenic chemicals. The measuring technique is based on the in vitro activation of the microsomal enzyme, biphenyl-2-hydroxylase. It showed that carcinogenic chemicals such as benzpyrene, aflatoxin, and β-naphthylamine produced substantial increases in the 2-hydroxylation of biphenyl with no similar increases in the 4-hydroxylation of this model substrate. Knowledge concerning drug metabolism may often point to the potential toxicity of the compound, such as the possible carcinogenicity and hemolytic activity of hydroxylamines formed in the N-hydroxylation of amines and the reduction of aromatic nitro compounds, and the potential hepatotoxicity of compounds containing the allyl group due to the destruction of cytochrome P-450. Similarly, a knowledge of the kinetics of distribution of the drug and its metabolites correlated with the pharmacological activity is invaluable in selection of the most appropriate animal species for toxicity and pharmacology studies, in calculation of appropriate human dosage, and in monitoring of safety and efficacy in humans. (39 references)

3005 POTENTIAL CARCINOGENICITY OF FOOD ADDITIVES AND CONTAMINANTS. (Eng.) Shubik, P. (Univ. Nebraska Medical Center, Omaha, NB 68105). *Cancer Res* 35(11/Part 2): 3475-3480; 1975.

The potential role in carcinogenesis of food contaminants and additives presents a complex problem in assessing the risk to the public. Long-term testing in laboratory animals is still the most feasible method for determining potential carcinogenicity of various chemicals. The disadvantages encountered in the present methods of animal testing are discussed, and a review is made of the current status of particular food contaminants and additives (under scrutiny as possible carcinogens). Since it may not be possible to remove all carcinogenic materials from the environment, methods to neutralize or mitigate their harmful effects should be sought. Greater cooperation is called for among food technologists, toxicologists, laboratory researchers, and epidemiologists in the decision-making process regarding the role of possibly carcinogenic contaminants and additives. (20 references)

3006 DIET AND THE CHEMICAL ENVIRONMENT AS MODIFIERS OF CARCINOGENESIS. (Eng.) McLean, A. E. M. In: *Host Environment Interactions in the Etiology of Cancer in Man. Proceedings of a Meeting Held at Primosten, Yugoslavia, 27 August-2 September 1972.* International Agency for Res. on Cancer and John E. Fogarty International Center of the Natl. Insts. of Health USA. (Lyon, France): pp. 223-230; 1973.

Studies on diet and the chemical environment are reviewed and discussed with regard to their relation to carcinogenesis. Studies of migrant peoples suggest that the differing patterns of cancer incidence are mostly determined by environmental rather than genetic factors. Microsomal hydroxylation seems to be responsible for the activation of polycyclic hydrocarbons, carcinogenic amines, pyrrolizidine alkaloids, nitrosamines, and aflatoxin in the body. The interaction between carcinogen and the target animal depends on the exact level of hydroxylation enzymes in the animal, and in some instances one level of hydroxylation will be carcinogenic while a higher level will protect animals from toxic or carcinogenic action. Dietary factors alter microsomal hydroxylation activity in many tissues in experimental animals. Because populations differ widely in their dietary habits in ways that may alter their microsomal hydroxylation activity, methods must be developed to measure microsomal hydroxylation in large numbers of people in order to see whether patterns of hydroxylation correlate with cancer patterns. (45 references)

3007 POSSIBLE ROLE OF NUTRIENTS IN NEOPLASIA. (Eng.) Vitale, J. J. (Boston Univ. Sch. Medicine, Boston, MA). *Cancer Res* 35(11/Part 2): 3320-3325; 1975.

The relationship between cancer and diet is supported by epidemiological evidence, which suggests that diet is a factor in the development of cancer of various organs (including the esophagus, stomach, liver, and large bowel). The role of depressed immunocompetency is stressed as a possible important consequence of nutritional insults leading to carcinoma. Study of the relationships among nutritional factors, host-defense mechanisms, and carcinogens to carcinogenesis is significant because, theoretically, manipulation of diet could suppress or prevent chemically induced cancers. The distinction should be emphasized, however, between ''nutrients,'' those elements of one's intake that are essential because the body is unable to synthesize them de novo, and ''diet'', which refers to everything that is ingested. It is the components of diet (chemicals in particular) that are most convincingly implicated as carcinogens; it is doubtful that nutrient deficiencies or excesses have a direct causal relationship with the onset or development of cancer. Rather, nutrition probably exerts its carcinogenic effects indirectly; nutrients possibly enhance chemical carcinogenesis multifactorially. (57 references)

3008 NUTRITION AND EXPERIMENTAL CARCINOGENESIS: A REVIEW. (Eng.) Clayson, D. B. (Univ. Nebraska Medical Center, Omaha, NB 68105). *Cancer Res* 35(11/Part 2): 3292-3300; 1975.

The restriction of total diet or number of calories fed to rats and mice inhibits the formation of tumors in several tissues. Unless the animals are fed equivalent levels of food, or attain equivalent body wt, it is difficult to assess the significance of the effect of other nutritional modifications on carcinogenesis. The effects of altering the levels of protein or fat are less than those seen with dietary restriction. Feeding a protein-free diet is tolerated for a limited period and can alter the metabolism of carcinogens. It may affect the tumor incidence induced by one-shot carcinogens. Vitamins have specific effects on the activity of certain carcinogens, the fullest information being available for vitamin A (which has been shown to inhibit or enhance carcinogenesis) and vitamin C (which by reducing sodium nitrite, prevents nitrosation of secondary and tertiary amines occurring in acidic conditions of the stomach). Inorganic substances, such as iodine (thyroid) and copper (liver), may affect the tumor incidence in specific tissues. The metabolic activation of carcinogens is modified by enzyme induction and the administration of antioxidants. (114 references)

3009 INTERACTIONS OF BACTERIA AND DIET IN THE ETIOLOGY OF CANCER. (Eng.) Hill, M. J.; Williams, R. E. O. In: *Host Environment Interactions in the Etiology of Cancer in Man. Proceedings of a Meeting Held at Primosten, Yugoslavia, 27 August-2 September 1972.* International Agency for Res. on Cancer and John E. Fogarty International Center of the Natl. Insts. of Health USA. (Lyon, France): pp. 231-235; 1973.

The idea that diet may generate cancer through its influence on, or by providing a substrate for, bacteria that produce a carcinogen is discussed. Both nitrate- and nonnitrate-reducing enterobacteria can promote the nitrosation of secondary amines, although the latter require nitrite while the former can also use nitrate as the substrate. The simplest situation in which carcinogens may be produced is that in which microbes are present in a foodstuff that also contains the substrates for nitrosation; this is illustrated by a report on nitrosamines produced during the drying of fish in which the fish, with intestines intact, are treated with sea salt containing nitrate as a contaminant. Carcinogens may also be formed when one or both substrates are present in the food but the bacteria are those already present in the body. The stomach would seem the most likely place for the interaction to occur, with nitrosodimethylamine being the nitrosamine most likely to be formed. In addition to nitrosamines, bacteria can also produce, from biliary steroids, deoxycholic acid, estradiol, and the 17-methoxy derivative of estradiol, which also may be carcinogenic. (25 references)

3010 FUTURE INQUIRIES INTO THE EPIDEMIOLOGY OF GASTRIC CANCER. (Eng.) Graham, S. (Dept. Social and Preventive Medicine, State Univ. New York, Amherst, NY 14226). *Cancer Res* 35(11/Part 2): 3464-3468; 1975.

Since gastric cancer has been shown in many studies to be strongly related to a large number of social factors, ethnic background, occupation, and socioeconomic status, the importance of exogenous factors in its etiology is suggested. Past studies show a variety of relationships that furnish leads requiring elucidation to further understanding of this disease, such as higher risks related to exposures to asbestos, metals, dusts, purgatives, and poor dental hygiene. Diet inquiries have steadily improved, and negative relationships have been discovered with ingestion of vegetables in studies in Wales, Liverpool, Buffalo, Honolulu, Japan, and Norway. Positive relationships with starch have been found in many of these same places as well as in Israel. These leads need further investigation (as do better methods of diet research, studies of diet and histological type of gastric cancer, diet in its relationship to familial aggregation of gastric cancer, and studies of consistencies in relationships among the various sites in the digestive tract). Since diet is so heterogeneous in modern societies, multivariate analysis is needed. (27 references)

3011 DEVELOPMENTS IN THE EPIDEMIOLOGY OF STOMACH CANCER OVER THE PAST DECADE. (Eng.) Haenszel, W. (Natl. Cancer Inst., Bethesda, MD 20014); Correa, P. *Cancer Res* 35(11/Part 2): 3452-3459; 1975.

History of stomach cancer epidemiology is reviewed. The introduction of migrant population studies in the 1960 decade describing the critical role of exposures to this disease in early life is a key event. Companion pathology studies indicate different epidemiological patterns for histological entities, intestinal and diffuse type carcinomas, and confirm an excess of intestinal metaplasia in populations at high risk to stomach cancer. Recent results suggest that epidemiology of stomach cancer can be transformed into the epidemiology of precursor lesions, and introduction of the fiber optic gastroscope makes technically feasible detailed studies of the relationship of precursor lesions to suspect factors, including diet, in selected geographic areas. Nitroso compounds have been identified as candidate carcinogens, and the epidemiological, pathological, and chemical data display signs of internal consistency. Feeding experiments with N-methyl-N'-nitro-N-nitrosoguanidine lead to animal models permitting a coordinated epidemiological-experimental approach to stomach cancer. (40 references)

3012 ANIMAL MODELS FOR THE STUDY OF DIETARY FACTORS AND CANCER OF THE LARGE BOWEL. (Eng.) Reddy, B. S. (Naylor Dana Inst. for Disease Prevention, American Health Foundation, Valhalla, NY 10595); Narisawa, T.; Maronpot, R.; Weisburger, J. H.; Wynder, E. L. *Cancer Res* 35(11/Part 2): 3421-3426; 1975.

The studies in metabolic epidemiology show a strong association between dietary fat intake, level of fecal anaerobic bacteria, fecal acid, and neutral sterols and the risk of colon cancer among different populations. Current concepts visualize that colonic bile acids and cholesterol metabolites play a modifying role in large bowel carcinogenesis; these compounds are derived from dietary factors (directly or indirectly), and they subsequently are modified by the intestinal bacteria. In the animal model, two bile acids (lithocholic and taurodeoxycholic) act as colon tumor promoters. Rats fed a high-fat diet are more susceptible to colon tumor induction by 1,2-dimethylhydrazine compared to animals fed a normal-fat diet. The intestinal microflora also play a modifying role in enhancing colon tumor production by 1,2-dimethylhydrazine. (18 references)

3013 CASE STUDY 5: ALDRINE AND DIELDRIN SUSPENSION BASED ON EXPERIMENTAL EVIDENCE AND EVALUATION AND SOCIETAL NEEDS. (Eng.) Epstein, S. S. (Case Western Reserve Medical Sch., Cleveland, OH 44106). *Ann NY Acad Sci* 271: 187-195; 1976.

The carcinogenicity of dieldrin has been unequivocally demonstrated in independent studies using several strains of mice. These data largely relate to hepatocarcinogenicity, but some evidence of extraphepatic multiple site neoplasms has also been found. Dieldrin was carcinogenic at the lowest dosage tested, 0.1 ppm. More limited studies in the rat clearly indicate that dieldrin is carcinogenic; one study demonstrated the induction of liver cancers and of multiple site tumors. Other studies in rats, monkeys, and dogs were rejected for methodological inadequacies. Limited industry studies of occupationally exposed adult males also point to its carcinogenicity. The experts are unanimous in that the data accumulated so far cannot validate the noncarcinogenicity of dieldrin. As dieldrin exposure is involuntary, and as it constitutes a widespread contaminant of food, air, and water, it is felt that dieldrin presents an imminent hazard to exposed populations. (27 references)

3014 GENERAL INFORMATION AND CONCLUSIONS ON PYRROLIZIDINE ALKALOIDS. (Eng.) IARC Working Group. In: *IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Man: Some Naturally Occurring Substances. Meeting Held in Lyon, 7-13 October 1975.* International Agency for Res. on Cancer. (Lyon, France): vol. 10, pp. 333-343; 1976.

The following are reviewed briefly: toxicity and carcinogenicity studies of plants and plant extracts containing pyrrolizidine alkaloids and alkaloid mixtures; general methods of analysis for pyrrolizidine alkaloids; their general metabolism and hepatotoxicity; and the carcinogenicity of metabolites. About 30 pyrrolizidine alkaloids have been found to be hepatotoxic, mostly in rodents, and circumstantial evidence suggests that some are hepatotoxic to man. Evidence from animal studies suggests that the hepatotoxicity is due to the formation of toxic pyrrolic metabolites by the mixed function oxidases of liver cells. The initial product formed from alkaloids that are esters of heliotridine (e.g., lasiocarpine) or retronecine (e.g., jacobine, monocrotaline, retrorsine, riddelliine, and seneciphylline) is very probably the dehydroalkaloid. Alkaloids that are esters of otonecine (e.g., hydroxysenkirkine and senkirkine) are converted into the dehydroalkaloid dehydroretronecine via demethylation. The dehydroalkaloids are highly reactive alkylating agents: dehydroretronecine is carcinogenic in rats. Local rhabdomyosarcomas developed in 31/60 young male Sprague-Dawley rats given 20 mg/kg twice weekly for 4 mo sc then 10 mg/kg for 8 mo sc and killed when moribund over the next 10 mo. Carcinogenicity tests should be undertaken on all untested hepatotoxic pyrrolizidine alkaloids to which humans may be exposed. This exposure may arise by the intentional use of certain herbs as remedies for ailments or as food, or unintentionally, by the contamination of food crops by alkaloid-containing plants. (42 references)

3015 HEALTH PROBLEMS ASSOCIATED WITH NITRITES AND NITROSAMINES. (Eng.) Lijinsky, W. (Oak Ridge Natl. Lab., PO Box Y, Oak Ridge, TN 37830). *Ambio* 5(2): 67-72; 1976.

The seemingly ubiquitous health problems associated with nitrites and nitrosamines are discussed at length. N-nitrosamines are the most widespread and potent carcinogens that humans contact. Nitrites are encountered naturally as residues on plants, preservatives of meats, and necessary ingredients in many drugs. The amino acids present in meats allow the natural conversion of nitrites to nitrosamines; the stomach produces the largest amounts of the latter. Nitrosamines have been shown to act additively and synergistically, making an estimation of risk almost impossible. The detection of the carcinogen is difficult, and as a result the exposure rate for humans has not been quantified. It is concluded that nitrites standards in the food and drug industry should be reduced to the minimum consistent with safety and achievable by good manufacturing procedures; these would be lower than those currently allowed. Failing this, the public should be educated to the dangers of products with high nitrite concentrations so that they can reduce or eliminate their consumption. (28 references)

3016 PUBLIC-HEALTH ROUNDS AT THE HARVARD SCHOOL OF PUBLIC HEALTH. VINYL CHLORIDE: CAN THE WORKER BE PROTECTED? (Eng.) Wegman, D. H. (665 Huntington Ave., Boston, MA 02115); Peters, J. M.; Jaeger, R. J.; Burgess, W. A.; Boden, L. I. *N Engl J Med* 294(12): 653-675; 1976.

The toxicology, economic and engineering aspects, and regulation of occupational exposure to vinyl chloride are reviewed. Well before 1974, when three cases of a rare neoplasm, angiosarcoma of the liver, were reported in workers exposed to vinyl chloride over a 20-yr period, there was sufficient evidence to support the presumption of a serious occupational hazard. Toxicological studies as early as 1938 indicated that vinyl chloride has little acute lethal action and negligible short-term toxicity but is a potent carcinogen in rodents under worklike exposure of 50 ppm and less. Useful toxicologic screening should be carried out in locations other than the workplace and in species other than man. The manner in which workers are exposed to vinyl chloride varies greatly, depending on their occupation in one of three stages: (a) production of vinyl chloride monomer (VCM); (b) reaction of VCM to form polyvinyl chloride (PVC); and (c) the fabrication of plastic products from PVC. Major exposure and the cases of angiosarcoma of the liver occur in the second industrial stage. The most common exposure in the first stage occurs from leaks and faulty equipment maintenance. In the third stage, exposure (1,000 ppm) occurs when workers periodically crawl into the reactor to clean the inside surface. The Federal occupational standard for vinyl chloride exposure (issued in October 1974), although accepting in principle 1 ppm as the maximum possible exposure, permits temporary exposures of up to 25 ppm without respiratory protection. (27 references)

3017 CIGARETTE SMOKING AND LUNG DISEASES: AN UPDATE. (Eng.) Ayres, S. M. (St. Louis Univ. Sch. Medicine, St. Louis, MO). *Respir Care* 21(7): 632-640; 1976.

Almost all pulmonary disease is related to the inhalation of infectious or irritant particulate material. Carcinoma of the lung and cigarette smoking have been strongly linked epidemiologically. Cigarette smoking has also been closely linked to the conditions that are grouped together as chronic obstructive lung disease. Ventilation/perfusion abnormalities appear in smokers with small airway involvement, but overall resistance patterns are often normal. An autopsy study revealed that 40% of smoking men who were presumed healthy had grade 20 emphysema, as opposed to 10% of nonsmoking men. All individuals exposed to the same amount of cigarette smoke do not exhibit identical manifestations. The highest incidence of lung disease is found in individuals with a genetic predisposition and who smoke. Data on the pollutant-cigarette interaction hypothesis suggest a true synergism between cigarette smoking and certain occupational and community pollutants such as asbestos, uranium, chromium, nickel, and arsenic. Cessation of smoking appears to be related to decrease in mortality due to carcinoma and lung disease. Mortality rates for British physicians who stopped smoking decreased by 24% for obstructive lung disease and by 38% for carcinoma of the lung from 1961 to 1965. Passive smoking, which is exposure to cigarette smoke, has been associated with angina pectoris, an increase in blood carboxyhemoglobin, and with pneumonia and bronchitis in the first yr of life. (45 references)

3018 PHYSIOLOGICAL EFFECTS AND METABOLISM OF GOSSYPOL.

(Eng.) Abou-Donia, M. B. (Duke Univ. Medical Center, Durham, NC 27710). *Residue Rev* 61: 126-160; 1976.

The biological and physiological effects of gossypol, a yellow coloring matter from various parts of the cotton plant, on several species are discussed, and its metabolism, chemistry, and biosynthesis are reviewed. Antitumor activity (40%-60% inhibition) against Ehrlich's ascites carcinoma strain C has been reported after sc injection of relatively large doses, but it was inactive when administered po. Toxicity in nonruminant animals may be acute (circulatory failure), subacute (pulmonary edema), or chronic (symptoms of malnutrition and ill health). Lack of toxicity (resulting from protein binding) in ruminants has been attributed to prolonged mastication, water content, and increased time in the lumen. A number of biochemical effects have been reported, including the uncoupling of oxidative phosphorylation in rat liver mitochondria in vitro. In human feeding experiments, no ill effects of incorporation of cotton seed oil into the diet have been reported. (200 references)

3019 THE ESTROGEN-CANCER HYPOTHESIS.

(Eng.) Hertz, R. (George Washington Univ. Sch. Medicine, 2300 Eye St. N.W., Washington, DC 20037). *Cancer* [*Suppl*] 38(1): 534-540; 1976.

The estrogen-cancer hypothesis postulates that both exogenous and endogenous estrogens and their metabolites play a significant and possibly an etiologic role in the pathogenesis of cancer in the estrogen-responsive tissues of man and animals. Laboratory experience has indicated that estrogen administration is followed by regularly reproducible tumors of the breast, cervix, endometrium, pituitary, testicle, kidney, and bone marrow in mice, rats, rabbits, hamsters, and dogs. In human exposure, a significant impact might be expected from dosage levels, age and chronicity of exposure, ethnic or genetic derivation, and the potential presence of predisposing viruslike factors affecting the ultimate hormone response. The genetic factor seems most patently applicable to man, as there is an increased risk of breast cancer to daughters of affected women. In relation to dosage, no extrapolation can be made from the animal data in general; but a chronic elevation above the level of endogenous estrogen production would qualify as a potential carcinogenic dose. Highly conclusive reports from several sources have recently demonstrated an association between maternal exposure to estrogens during pregnancy and the subsequent incidence of vaginal adenocarcinoma in the female children. The paradoxical effectiveness of massive estrogen doses in inducing remissions in older women with inoperable breast cancer remains an enigmatic phenomenon. However, there is an equally impressive body of data relating endogenous estrogen to cancer of the breast and endometrium. (49 references)

3020 THE INDUCTION OF TUMOURS BY HORMONES.

(Eng.) Muhlbock, O. (Netherlands Cancer Inst., Amsterdam, The Netherlands). *Int Union Cancer Tech Rep Ser* 20: 89-93; 1975.

The interrelationships between hormones and the occurrence of malignant tumors is reviewed. The hormones involved are mainly the growth-stimulating hormones each acting on a particular target organ. Tumors of the thyroid can be induced by the thyrotropic hormone, tumors of the adrenal cortex by the adrenocorticotropic hormone, and certain types of tumors of the ovary and the testis by gonadotropic hormones. It appears, therefore, that the group of hormones with a carcinogenic effect includes primarily the tropic hormones of the hypophysis and the steroid hormones. Other hormones, such as insulin or thyroxine, which are not known to be carcinogenic, generally act on the metabolism without a special target organ. An exception to this generalization is growth hormone or the somatotropic hormone of the hypophysis, which is a metabolic hormone with no specific target organ. Growth hormone may act as an accelerator of tumor development rather than as a causative factor. Generally, the carcinogenic effect of a hormone is related to the dose of the hormone acting on its target organ. When given a dose of estrogenic hormone at the physiological level, castrated male mice develop only a very few mammary tumors and only very late in life. With a dose 16 times higher, however, nearly all animals develop mammary tumors very early in life, indicating the dose-response effect of the hormone. Another usual characteristic of hormonal induction of tumors is that a tumor will develop only if the organ is under a continuous growth-stimulating influence. When one group of gonadectomized mice was treated with estrogenic hormone continuously and a second group with double the amount intermittently, only the group with continuous treatment had a high incidence of mammary carcinomas. Continuous growth-stimulating effect by a hormone can lead to malignant neoplasia in the stimulated organ. (6 references)

3021 CONCORDE AND SKIN CANCER.

(Fre.) Denisse, J. F. (Observatoire de Meudon, 92130 Meudon, France). *Biomedicine* 24(1): 16-17; 1976.

A long chain of ill-understood facts stands between the Concorde flights and a possible increase in skin cancer. Supersonic planes (SST) must travel at an altitude of about 15,000 m, at which they meet little resistance in a rarefied atmosphere. This environment, the stratosphere, is stable, without any precipitation or much turbulence and, therefore, renews itself very little or very slowly. Some fear that the SST would contribute to the transformation of the stratosphere into a vast ''trash can.'' The accumulation of industrial waste, they contend, may in time affect the transparence of the sky and the radiation of the sun and the Earth, shattering the equilibrium that controls our climate. Foreign bodies, even traces, acting like catalysts, could upset the chemical balance regulating the composition of the stratospheric milieu. This would cause a diminution of ozone, which could result in a greater filtration of UV rays. Plane engines, at high temperatures, eject large quantities of wastes, especially nitrogen oxides. These gases could remain long enough in the stratosphere to modify the ozone content. Very little is known about how long the pollutants stay in the stratosphere or about the nature of their action on the ozone. Dome calculations indicate that the activity of a fleet of 100 Concordes would result in a 1% reduction of the ozone concentration. This must be compared to the natural variations in the ozone, 10% from one month to the next, 50% according to season or latitude. Also, the amount of nitrogen oxides from 100 Concordes would remain below their natural accumulation. It is believed that a reduction in ozone by any percentage would, at worst, increase by a double percentage the incidence of skin cancers, epitheliomas, or melanomas. These cancers, in France, account for only a few hundred deaths yearly. So, the flights of 100 Concords might mean, for the French population (51 million), 10 additional skin cancers every year. This seems negligible when compared to the many other dangers to life that are willingly accepted. (No references)

3022 COMPARISON OF ALKYLATING AGENT AND RADIATION CARCINOGENESIS: SOME ASPECTS OF THE POSSIBLE INVOLVEMENT OF EFFECTS ON DNA. (Eng.) Lawley, P. D. In: *Biology of Radiation Carcinogenesis*. Edited by J. M. Yuhas, R. W. Tennant, and J. D. Regan. (New York: Raven Press): pp. 165-174; 1976.

The possible significance of the extent of chemical modification wrought on DNA by isotopically labeled carcinogens was discerned by comparison of the effects of chemical carcinogens on DNA with those of radiation. A series of alkylating agents was classified in terms of increasing relative ability to react at O-atom sites in DNA: methyl methanesulfonate, ethyl methanesulfonate, N-methyl-N-nitrosourea, and N-ethyl-N-nitrosourea. This order of increasing ability to induce reversion mutations, probably GC=AT transitions, in phage T4, agrees with the concept that O^6-alkylguanines are directly miscoding bases. This series of agents was also used to study comparative carcinogenicity with respect to x-ray induction of thymic lymphoma in mice. A positive correlation between ability of agents to alkylate the O-6 of guanine in thymic DNA and carcinogenic potency was found. Although methyl methanesulfonate was not active in this system, it can induce tumors in other systems. The methylating agents induce single-strand breaks in DNA, and the principal repair system appears to fall into the category of ''short'' repair. These single-strand breaks may result from spontaneous hydrolytic depurinations of 3- and 7-methylpurines or from enzymatic depurinations (e.g., 3-alkyladenine and O^6-alkylguanine). Arylkylating agents, which are also carcinogens, can evoke an alternative repair response of the *uvr* type. (40 references)

3023 RADIATION CARCINOGENESIS. (Eng.) Hirohata, T. (Sch. Public Health and Cancer Center, Univ. Hawaii, 1960 East-West Road, Honolulu, HI 96822). *Semin Oncol* 3(1): 25-34; 1976.

Evidence linking exposure of human populations to radiation to an increased risk of cancer is summarized. An advantage in the study of radiation carcinogenesis is that time and dose of exposure are often easy to ascertain. Therapeutic irradiation has been used to treat conditions such as ankylosing spondylitis, metropathia hemorrhagica, and other benign gynecological disorders; thymic enlargement in infants; and tinea capitis. Most treatments resulted in an increase in leukemia that could be observed after several years and an increase in tumors at particularly heavily irradiated sites after longer periods of latency. As to leukemia, a study of 14,500 spondylitis patients treated with x-ray irradiation to their spines demonstrated an increased mortality with increasing dose, with a slope of 0.5 case/10^6/yr/rad. Diagnostic irradiation has also resulted in an increased risk of cancers, and tuberculosis patients exposed to repeated fluoroscopic examinations were found to have an increased risk of breast cancer. Prenatal irradiation is associated with an excess risk of leukemia: the maximum likelihood of excess risk was estimated at 1.4 times that of nonirradiated children. Long-term surveillance of 100,000 atom bomb survivors at Hiroshima and Nagasaki demonstrated an increase of leukemia incidence with increasing estimated air radiation dose, with values of 3 and 1.6 cases/10^6/yr/rad at Hiroshima and Nagasaki, respectively, for the period 1950-1966. The higher incidence at Hiroshima may be related to the high relative biological effectiveness of the neutrons, to which the subjects were exposed together with gamma rays. The survivors at Nagasaki were exposed predominantly to gamma rays only. It became evident nearly 20 yr after exposure that the survivors also had excessive mortality from cancers at other sites, primarily in the respiratory tract and breast. (60 references)

3024 THE REGULATION OF ERYTHROPOIESIS. (Eng.) Rifkind, R. A. (Coll. Physicians and Surgeons, Columbia Univ., New York, NY 10032); Marks, P. A. *Blood Cells* 1(3): 417-428; 1975.

Studies on the proliferation and differentiation of erythroid precursor cells in fetal mouse liver cells and Friend virus-transformed murine erythroleukemia cells (MELC) are reviewed. Stimulation of RNA synthesis is the earliest detected effect of erythropoietin both in fetal and adult marrow erythroblasts. An increase in the RNA synthesis rate is detected in cultured fetal mouse cells within 1 hr of exposure to erythropoietin. Biologically active globin mRNA is not detected until 5-10 hr of culture with the hormone, indicating that mRNA is produced later than the initial burst of hormone-mediated ribosomal and transfer RNA synthesis. Molecular hybridization experiments suggest that the primary effect of erythropoietin is on erythroid precursor cell proliferation and not on the rate of accumulation of differentiation-specific products such as globin or globin mRNA. A hypothetical model is given for the regulator sites in erythropoiesis: an early level of regulation occurs at the recruitment of pluripotent stem cells in the erythropoietin-sensitive cell compartment. Another major locus is the proliferation of early erythroid precursors; erythropoietin is implicated in this step. The requirement of erythropoietin for proliferation of the erythroid precursor cells appears to be obviated in MELC cultured in dimethylsulfoxide. The effect of highly polar compounds, such as dimethylsulfoxide, appears to be to accelerate the rate of spontaneous erythropoietic differentiation. This process may be mediated by an effect of these compounds on the configuration of chromatin and the transcription of specific RNA products required for the initiation of erythroid cell differentiation. (31 references)

3025 PROPERTIES OF THE MOUSE MAMMARY TUMOUR VIRUS AND OF PARTICLES FOUND IN HUMAN MILK. (Eng.) Moore, D. H.; Sarkar, N. H.; Dion, A. S.; Charney, J.; Lasfargues, E. Y.; Kramarsky, B. In: *Host Environment Interactions in the Etiology of Cancer in Man. Proceedings of a Meeting Held at Primosten, Yugoslavia, 27 August-2 September 1972.* International Agency for Res. on Cancer (Lyon, France): pp. 377-388; 1973.

Research concerning properties of the murine mammary tumor virus (MuMTV) and particles found in human milk is reviewed. MuMTV virions can be separated in relatively pure form by diluting skimmed mouse milk with buffer and solubilizing the casein with EDTA. Pelletized virus is resuspended and laid over a continuous gradient of Ficoll or sucrose; a large percentage of the virions form a recoverable band. Electron microscopy showed that the virus has a tendency to adhere to other particles and to form aggregates with itself. The pH mobility curve indicated an isoelectric point at pH 2.5; bioactivity showed little falling off between pH 5 and 11. The MuMTV virion has a characteristic membrane with protrusions extending from its outer surface. Viral RNA filaments showed length distribution peaks at 1.2, 2.4, and 3.6 μm; the longest molecule had an estimated molecular wt of 3.6×10^6 daltons, although sedimentation analysis of extracted RNA indicated a molecular wt of 10×10^6 daltons. Treatment of the MuMTV virion with Tween 80 and ether resulted in disruption of the viral membrane and release of the nucleoid, large fragments of membrane, and short pieces of membrane that appeared as rosettes. Screening a variety of neoplasms by immunodiffusion and immunofluorescence tests demonstrated MuMTV infection in mammary tumors, ML leukemias, and Leydig cell tumors of MuMTV-infected mice. The following evidence indicates that human breast cancer and the putative virus have some properties in common: in both mouse and man there is a high mammary tumor incidence in particular family groups: particles have been found in human milk that are morphologically similar to MuMTV virions; some human sera have a neutralizing effect on MuMTV; both mouse and human milk contain particle-bound 60S-70S RNA and a resident RNA-templated DNA polymerase; nucleic acid hybridization between mouse virus products and human mammary tumor products has been observed. In addition, in both mouse and man the onset of the tumors occurs at two different periods in life. (31 references)

3026 THE ROUS SARCOMA VIRUS STORY: CYTOGENETICS OF TUMORS INDUCED BY RSV. (Eng.) Mitelman, F. In: *Chromosomes and Cancer.* Edited by J. German. (New York: John Wiley & Sons): pp. 675-693; 1974.

The effects of Rous sarcoma virus (RSV) on chromosomes in vivo were studied with cells from primary, metastatic, and transplanted tumors induced in the mouse, Chinese hamster, and the rat. Striking similarities are seen in the general numerical pattern of the chromosomes in RSV-Schmidt-Ruppin-induced sarcomas from different species. It was suggested that all neoplasms are initially composed of normal diploid cells and that, with increasing age of the tumor, these cells decrease in number at an accelerated rate. The initial deviation from normality is probably the formation of a trisomic karyotype, judging from the observation that trisomy was the dominant abnormality in all three species. The results suggest a specificity in the participation of the genetic apparatus of the host cell in the early stages of oncogenesis; with different oncogenic agents, different molecular pathways might be involved. Tumors within a well-defined histopathologic category could actually consist of cytogenetically different subgroups, each determined by its mode of induction. The apparent lack of consistent karyotypic abnormalities in most malignancies might be partly explained by assuming that tumors in the same histopathological category can be produced by different etiological agents. (45 references)

3027 EPIDEMIOLOGICAL EVIDENCE FOR THE ROLE OF A VIRUS IN THE ETIOLOGY OF TUMOURS. (Eng.) Day, N. E. In: *Host Environment Interactions in the Etiology of Cancer in Man. Proceedings of a Meeting Held at Primosten, Yugoslavia, 27 August-2 September 1972.* International Agency for Res. on Cancer (Lyon, France): pp. 367-375; 1973.

The types of epidemiological data used as evidence of a viral etiology of cancer are reviewed. Evidence for case-to-case transmission (as in cervical cancer), clustering of cases (as in Burkitt's lymphoma), vertical transmission (as in breast cancer), and for increased risks due to immune deficiency, geographical variations, and age distribution is discussed. Relationships between specific virus infections, such as Epstein-Barr virus, and neoplastic diseases are considered. The strongest evidence for this association comes from case-control comparisons. Population comparisons on a larger scale have also been advanced. Caution is recommended because a virus may be a basic and necessary factor in tumor etiology, yet its oncogenic expression may be so thoroughly modified by other factors that it plays little part in risk differences between various groups. For example, the main geographical variation in breast cancer incidence is clearly due to a non-viral factor. Many people may be infected with an oncogenic virus without tumor induction, thereby diluting the comparison of infection prevalence between cancer cases and controls. In many populations prevalence may be high, but only in some groups will infection occur at the right time or in the presence of a synergistic cofactor. The most likely prospects for success in determining the role of virus infection in cancer etiology lie in identifying the specific conditions under which viral infection most fully realizes its oncogenic potential. Long-term prospective studies are needed. (47 references)

3028 IMPLICATIONS OF A VACCINE FOR THE PREVENTION OF EPSTEIN-BARR VIRUS INFECTION: ETHICAL AND LOGISTIC CONSIDERATIONS. (Eng.) Epstein, M. A. (Dept. Pathology, Univ. Bristol, Medical Sch., University Walk, Bristol BS8 1TD, England). *Cancer Res* 36(2): 711-714; 1976.

Evidence linking African Burkitt's lymphoma (BL) and nasopharyngeal carcinoma points to a causal role for Epstein-Barr (EB) virus in these cancers. It would seem that the only way to do this would be to develop a vaccine, vaccinate a population at risk in a high-tumor-incidence area, and subsequently follow the population for a consequential decrease in tumor incidence. Recent developments in the control of animal herpesvirus-induced malignant tumors by vaccines free of viral nucleic acid make it possible to envisage that a similar vaccine could be developed against EB virus without undue difficulty. Trial of the vaccine in human populations could be carried out by testing its ability to protect those at risk from primary EB virus infection accompanied by infectious mononucleosis. African BL provides uniquely favorable conditions in which to test for a causative role for EB virus: high incidence areas are known, the peak tumor incidence is at the age of 5 or 6, and the effects of vaccination on tumor incidence could be assessed within a decade. Should a carcinogenic role for EB virus be demonstrated in African BL, a program would be called for to extend the vaccine control of infection to areas where EB virus is implicated in the induction of nasopharyngeal carcinoma. A high incidence of this tumor is confined to populations of Southern Chinese origin, the very large numbers of such people and the frequency of the tumor among them make this a substantial world health problem and, therefore, worth the cost and effort necessary. (30 references)

3029 VIRAL LEUKAEMIA IN DOMESTIC CATS. (Eng.) Bennett, A. M. (Dept. Veterinary Pathology, Univ. Sydney, Sydney, N.S.W. 2006, Australia); Sabine, M. *Med J Aust* 2(2): 52-62; 1975.

The characteristics, immunological aspects, and epidemiology of viral leukemia in domestic cats are reviewed. A 5-yr survey revealed that one-third of feline malignant disease is hematopoietic. These leukemias are divided into lymphoreticular neoplasms and myeloproliferative disorders. The clinical course of both is short, with about 70% of animals dying within 8 wk of the onset of clinical signs. The first successful transmission of feline lymphosarcoma was achieved by inoculating neonatal kittens with a cell-free tumor extract derived from a naturally occurring thymic lymphosarcoma. The mature virion of the feline leukemia virus (FeLV) contains an inner electron-dense nucleoid representing the RNA genetic material and associated structural proteins. FeLV grows successfully in both monolayer and suspension culture. Antibodies detectable in the serum of many cats that have come into contact with FeLV or feline sarcoma virus appear to play an important part in limiting the development of virus-induced neoplasms. It is probable that both horizontal and vertical modes of transmission are involved in the spread of FeLV. Epidemiological and immunological studies have failed to show spread of FeLV to dog or man, although high doses of virus induce neoplasms in monkeys. (56 references)

3030 SOME NEW VIEWS OF THE CELL SURFACE. (Eng.) Edelman, G. M. (Rockefeller Univ., New York, NY 10021). *J Biochem (Tokyo)* 79(4): 1p-12p; 1976.

New investigational techniques in recent years have disclosed important new aspects of the surface of mammalian cells. The structure of the apparently ubiquitous histocompatibility antigens has been elucidated, and the dependence of lymphocyte/virus interactions on these antigens has suggested that histocompatibility molecules may serve as cell surface adaptors for the recognition of foreign antigens by T cell receptors. The free movement of constituents of the cell membrane, including histocompatibility antigens, in the bilayer of lipid that is the cell membrane has upset the old notion of fixed constituents. Cross-linking of cell surface proteins (e.g., antibodies) leads to clustering of these mobile receptor molecules into patches that are subsequently collected as a cap at one pole of the cell. Intercellular interactions and signaling from the surface of the cell to the interior depend on the structure and physicochemical properties of such receptors and on their linkage to structures underlying the cell membrane. An intracellular network of special proteins controls the motion and distribution of cell surface molecules. Mitogenic lectins such as concanavalin A bind and cross-link carbohydrate portions of surface receptors. This binding inhibits the motion of all other receptors on the surface. The nature of the controlling network appears to involve assemblies containing microfilaments and microtubules below the cell surface. Microtubules are important in transporting molecules out of the cell and in giving it shape. Microfilaments are related to components of muscle and are apparently involved in cell motion. Drugs preventing the assembly of microtubules block the signal induced by concanavalin A that commits the cell to divide. A connection between the network controlling cell mobility and the control of cell growth and division is postulated. It is suggested that the cell surface is under control of cytoplasmic structures and that these structures may be the information pathway that links events at the cell surface to the key control systems inside the cell that regulate motion, cell-cell interaction, and division. (28 references)

3031 THE CANCER CELL: DYNAMIC ASPECTS AND MODIFICATIONS IN CELL-SURFACE ORGANIZATION (FIRST OF TWO PARTS). (Eng.) Nicolson, G. L. (367 Steinhaus Hall, Univ. California, Irvine, CA 92717); Poste, G. *N Engl J Med* 295(4): 197-203; 1976.

Recent data have revealed a large number of surface differences between tumor cells and their normal counterparts. The topographic rearrangement of various cell surface components may be important in determining the altered surface properties and aberrant behavioral characteristics displayed by tumor cells. Various results and theories on trans-membrane control mechanisms have been incorporated into a unifying model that remains consistent with the data. Specifically, microtubule and microfilament assemblages are proposed to be interconnected and coupled to similar transmembrane anchorage points. These anchorage points could serve as regulatory centers for the organization of specific membrane domains. The contractile movements of the microfilament system would be opposed by an anchorage system made up of microtubules and would thus offer a system whereby the cell surface and its receptors would be under fine control by opposing membrane-associated cytoskeletal elements. (21 references)

3032 THE CANCER CELL: DYNAMIC ASPECTS AND MODIFICATIONS IN CELL-SURFACE ORGANIZATION (SECOND OF TWO PARTS). (Eng.) Nicolson, G. L. (367 Steinhaus Hall, Univ. California, Irvine, CA 92717); Poste, G. *N Engl J Med* 295(5): 253-258; 1976.

In reviewing aspects of the cell surface organization of cancer cells, one potentially important implication that emerges is that expression of certain surface alterations need not involve acquisition of uniquely ''tumor-specific'' surface components, but could equally arise simply from topographic rearrangement of existing surface components also present on normal cells. Another concept implicit in this proposal is that the topographic ''patterns'' of cell-surface molecules found in different cells could act as a ''code'' by which normal cells recognize each other and regulate their growth and social behavior and that alterations in such topographic patterns, if sustained, could result in the spectrum of aberrant behavioral characteristics displayed by cancer cells. Although a high degree of speculation surrounds this interpretation, sufficient experimental data are available to support a functional relation between the topography and dynamics of surface macromolecules and the control of cell surface properties. (50 references)

3033 HUMAN TUMOR XENOGRAFTS. (Eng.) Double, J. A. (Sch. Medicine, Univ. Leeds, Leeds LS29 NC, UK). *Biomedicine* 22(6): 461-465; 1975.

The use of human tumor xenografts as potential models for the study of human cancer and experimental chemotherapy is reviewed. Three systems are used for growing human tumor xenografts: (a) tumor tissue can be introduced into an immunologically privileged site where it remains free from attack by the host's immunological defense; (b) the hosts can be rendered immunologically incompetent so that they are unable to reject xenografts; and (c) xenografts can be grown in mutant nude mice that are genetically athymic and unable to reject foreign tumor grafts. If xenografts are to be used as models for chemotherapy, the basic rules built into protocols for assessing drug effects on transplantable tumors must be observed. Studies have shown that a bladder carcinoma, sensitive to 5-fluorouracil and x-rays and resistant to vinblastine, retained these characteristics when grown in immunosuppressed hamsters. In another study, the rank order of four drugs was the same in both species when the Walker rat fibrosarcoma was grown in immunosuppressed mice. If used correctly, the xenograft system could give clinically significant results of interest to immunologists, tumor biologists,- and pharmacologists. (34 references)

3034 THE LYMPHOID SYSTEM, IMMUNODEFICIENCY, AND MALIGNANCY. (Eng.) Good, R. A. (Memorial Sloan-Kettering Cancer Center, New York, NY 10021). *Adv Biosci* 12: 123-159; 1974.

The development of the immunological system is reviewed, and the association of both primary and secondary immunodeficiency and cancer is considered. T and B cell populations can be precisely quantified, and in healthy persons total nearly 100% of circulating lymphocytes. T cells are enumerated by: spontaneous rosette cell formation, specific anti-T cell cytotoxic antiserum, and scanning electron microscopy. B cells are enumerated by: immunofluorescence to quantify cells with readily demonstrable Ig at their surface, the presence of receptors for the Fc component of human IgG, rosette formation with human RBC based on Nussenzweig's C3 receptor, and scanning electron microscopy. In one form of immunodeficiency disease, in which IgG, IgA, IgM, IgD, and IgE are not present in the circulating blood, the patients often cannot make antibodies even after the most intensive and repeated antigenic stimulation. Many patients with x-linked infantile agammaglobulinemia seem to be completely lacking or almost completely lacking in B lymphocytes. These patients develop leukemia much more often than expected. Ataxia telangiectasia syndrome patients regularly have an immunodeficiency disease featuring deficits of cell-mediated immunities and a high frequency of malignancy. Autopsy material from these patients shows that fully 35% had malignant disease, 10% of which involved the lymphoreticular apparatus. Two kinds of malignancies seem to predominate in patients with common variable immunodeficiency disease: malignancies of the lymphoreticular system and gastrointestinal and colon carcinoma. Immunosuppressed patients have experienced inadvertent transplantation of cancer, but even widely disseminated cancer under such conditions can be eliminated completely immunologically. De novo malignancies occur more frequently than expected in immunosuppressed patients. Most are lymphoreticular and are not rapidly eliminated after cessation of immunosuppressive therapy. These findings seem to indicate that certain malignancies, particularly those of the lymphoreticular apparatus, are strongly favored by immunodeficiency. (80 references)

3035 THE HL-A SYSTEM AND NEOPLASIA. (Eng.) Lawler, S. D. In: *Cancer Genetics.* Edited by H. T. Lynch. (Springfield, IL: Charles C. Thomas): pp. 87-100; 1976.

The importance of the major histocompatibility complex system, HL-A, is discussed in relation to leukemia. Because of the relationship between the H2 system and virus-induced leukemia in mice, similar associations are sought in man. The worldwide data on Hodgkin's disease suggest a positive correlation with the presence of the W5 antigen and related antigens. Gestational choriocarcinoma is a malignant allograft. By typing of children antecedent to choriocarcinoma in those cases in which the tumor was preceded by a live birth, information about the HL-A constitution of the tumor can be obtained. Evidence was also obtained that choriocarcinoma is not preferentially associated with HL-A compatible conceptions; such conceptions differ from the mother over the whole range of HL-A specificities. It is suggested that the risk of a woman suffering from trophoblastic neoplasia after live-term birth, abortion, or pregnancy with a hydatidiform mole is not directly related to the choice of mate as far as the HL-A system is concerned. (56 references)

3036 IMMUNOLOGY, CANCER AND GENETICS. (Eng.) Chaperon, E. A. In: *Cancer Genetics.* Edited by H. T. Lynch. (Springfield, IL: Charles C. Thomas): pp. 52-62; 1976.

The application of immunological theory and techniques to the study of neoplastic diseases is reviewed. It is generally agreed that all tumors, at least at some time during their development, possess antigenic determinants that distinguish them from their hosts. Were it not for the surveillance activities of the immune system, the incidence of malignant disease would be far higher than it is. Data from animal experiments show a genetic basis, which may be linked to genes for histocompatibility antigens (HL-A), in the capacity of individuals to respond immunologically to certain antigens. Reports of high cancer incidence in some families and of the association of an increased frequency of certain HL-A antigens in individuals with a variety of malignant diseases may have some relation to this phenomenon. (72 references)

3037 MISCELLANEOUS PROBLEMS, CANCER, AND GENETICS. (Eng.) Lynch, H. T. In: *Cancer Genetics.* Edited by H. T. Lynch. (Springfield, IL: Charles C. Thomas): pp. 491-553; 1976.

Certain diseases or traits that appear to have a cancer association and show either a simple genetic inheritance pattern or appear to be familial, and developing areas of cancer research, are reviewed. The diseases are: cancer and collagen diseases, lupus-erythematosus; dermatomyositis; scleroderma; Sjogren's syndrome; childhood malignancies (surveyed epidemiologically); Wilms' tumor; rhabdomyosarcoma; neuroblastoma; hepatoblastoma; hepatocellular carcinoma; sacrococcygeal teratomas in children; Beckwith-Wiedemann syndrome; renal hamartomas, nephroblastomatosis, and fetal gigantism; Cowden's disease; Down's syndrome; renal carcinoma; testicular tumors; ovarian carcinoma; ovarian dysgerminoma; endometrial carcinoma; chondrosarcoma; osteogenic sarcoma; esophageal cancer; gastric cancer; gallbladder cancer; intraocular malignant melanoma (a genetic study of two families); carotid body tumors; Werner's syndrome; celiac disease; familial fibrocystic pulmonary dysplasia; nasopharyngeal carcinoma; hereditary multiple exostosis; thymoma; metastasizing carcinoid tumors; α_1 antitrypsin deficiency and hepatoma; Poland's syndrome; hereditary bone dysplasia with sarcomatous degeneration; myxoma of the heart; X-linked recessive reticuloendothelial syndrome with hyperglobulinemia; urinary bladder cancer; carcinoma of the ureter; carcinoma of the larynx; genetics and virilizing adrenal carcinoma; leukocytic mucopolysaccharides in cancer patients; Burkitt's lymphoma; arrhenoblastoma; infantile genetic agranulocytosis; hereditary pancreatitis; cancer of the prostate; multiple myeloma; and C-monosomy myeloproliferative syndrome. (348 references)

3038 CHROMOSOMES IN CLINICAL ONCOLOGY. (Eng.) Sandberg, A. A. In: *Perspectives in Cancer Research and Treatment. Contributions in Honor of the Seventy-Fifth Anniversary of the Roswell Park Memorial Institute.* (New York: Alan R. Liss, Inc.): pp. 223-236; 1973.

The role of visibly recognizable karyotypic abnormalities in relation to the cause, diagnosis, therapy, and prognosis of human neoplasia is reviewed. There is no absolute evidence to date that such an abnormality, with the exception of the Philadelphia (Ph^1) chromosome, plays a direct role in the development of cancer or leukemia. Because about 80% of chronic myelocytic leukemia (CML) patients possess the Ph^1 chromosome, those that do not should perhaps be classified separately. Various syndromes with chromosome breakage show that not all such patients develop neoplasms, and where neoplasms with chromosome changes do occur, they differ from one subject to another. At least 50% of acute leukemia patients do not show karyotypic changes, demonstrating that such abnormalities are not necessary for the development of neoplasia in humans. When chromosomal changes do occur they are extremely diverse in nature, perhaps indicating that they are secondary to the neoplastic state. Although chromosomal changes have been reported in meningioma, generally considered a benign condition, their occurrence in any neoplasia implies an early or low malignancy for that tumor. Only very rarely have diploid human cancers been found; no two cancers have been shown to have an identical karyotypic makeup. In CML, a better response to therapy was noted when the Ph^1 chromosome was present. In acute myeloblastic leukemia the prognosis is poor if all the marrow cells have an aneuploid karyotype throughout the course of the disease. Although the Ph^1 chromosome is the only specific diagnostic abnormality found to date, the development of new techniques (such as fluorescent staining and the development of banding patterns) will probably yield further information. (6 references)

3039 MITOTIC ABNORMALITIES AND CANCER. (Eng.) Oksala, T.; Therman, E. In: *Chromosomes and Cancer*. Edited by J. German. (New York: John Wiley & Sons): pp. 239-263; 1974.

The mitotic aberrations that cause variations in the chromosome numbers of cancer cells are reviewed against the background of similar processes in nonmalignant cells. No single aberration has been found to be specific for cancer; however, in malignant tumors, abnormalities are not only greatly increased in frequency but seem to occur in characteristic combinations. Malignant tumors fall into two main classes: (a) the Ehrlich type (represented by the vast majority of cancers), in which the metaphase/prophase ratio (M/P) is increased; and (b) the rare Landschutz type, in which the M/P ratio is low. An increase in the M/P ratio reflects a longer metaphase or a shorter prophase, or both. At a certain point in the development of such tumors, multipolar divisions appear; the frequency of such divisions is positively correlated with malignancy. Positive correlations between increased M/P values and the occurrence of multipolar divisions is established; the correlation of a decreased M/P with the occurrence of endomitotic stages seems likely. No new completely cytological phenomena have been observed in cancer cells. Diagnosticians should be carefully trained in this area of cytology so that borderline cases can be diagnosed accurately. (63 references)

3040 IS THERE A ROLE FOR MITOCHONDRIAL GENES IN CARCINOGENESIS? (Eng.) Hoberman, H. D. (Albert Einstein Coll. Medicine, Bronx, NY 10461). *Cancer Res* 35(11/Part 2): 3332-3335; 1975.

Although defective respiration is not characteristic of all tumors, recent studies on the ultrastructure of normal and tumor cell mitochondria indicate that, in malignant cells, mitochondria deviate from normal not only in relative abundance but also in the size, form, density, and frequency of the appearance of lesions. Abnormal and normal mitochondria may populate the same cell. This suggests that there may be a gradation in respiratory deficiency depending on the proportion of normal to abnormal forms. Recent advances in mitochondrial genetics suggest that aberrant mitochondria may be formed as a result of the presence of an abnormal mitochondrial genome. In analogy with the petite mutant of certain strains of yeast, animal cells may be transformed by treatment with dyes that alter the structure of their mitochondrial DNA; their mitochondria also become deficient in enzymes of the respiratory chain. Whether nutritional or other deficiencies are mutagenic with respect to mitochondrial DNA of animal cells is not known. (37 references)

3041 THE ROLE OF CYTOGENETICS IN HEMATOLOGY. (Eng.) Rowley, J. D. (Dept. Medicine, Univ. Chicago, Box 420, Chicago, IL 60637). *Blood* 48(1): 1-7; 1976.

Chromosome analysis of bone marrow cells has provided new perspectives on the myeloproliferative syndromes and other hematologic disorders. Three different abnormal chromosome patterns have been observed in patients with refractory anemias: an additional No. 8 chromosome and deficiencies in the No. 5 and No. 20 chromosomes. Increased levels of glutathione reductase (GR) in RBC from two patients with sideroblastic anemia with an extra C (identified as No. 8) chromosome have been reported. A normal level of GR was found in patients with macrocytic anemia with a deficient No. 5 chromosome. In chronic myelogenous leukemia (CML), patients with the Ph^1 translocation have a much longer median survival than patients who are Ph^1-negative. A change in the karyotype of a Ph^1-positive patient strongly suggests that the patient is in the acute phase of CML. Patients with acute myelogenous leukemia with a normal karyotype have an 87% rate of induction of complete remission, whereas patients with an abnormal karyotype have a 20% remission rate. Chromosomal abnormalities observed in other hematologic disorders were reviewed briefly. (26 references)

3042 GENETIC AND TERATOGENIC ASPECTS OF WILMS' TUMOR. (Eng.) Strong, L. C. In: *Wilms' Tumor*: Edited by C. Pochedly and D. Miller. (New York: John Wiley & Sons); pp. 65-77; 1976.

Literature concerning the mutation model of Wilms' tumor and its association with congenital anomalies is reviewed. The development of Wilms' tumor is attributed to two mutational steps, the first of which may occur either in a germinal cell and result in hereditary Wilms' tumor or in a somatic cell and result in nonhereditary Wilms' tumor. Hereditary Wilms' tumor is transmitted as an autosomal dominant disorder with 63% penetrance, occurs at an early average age, and may be bilateral. Nonhereditary Wilms' tumor occurs at a later age and is single. The hereditary form of Wilms' tumor is occasionally associated with severe aniridia and may be related to a specific chromosomal deletion. The nonhereditary form of Wilms' tumor is occasionally associated with hemihypertrophy or genitourinary anomalies, suggesting some common control mechanisms during embryogenesis. Based on the recognition that all Wilms' tumors are not a homogeneous group etiologically, epidemiology studies may be designed to identify factors influencing the development of a specific subgroup of Wilms' tumors. Genetic counseling may be instituted regarding the risk to the affected individual of other tumors and the risk to the affected individual of other tumors and the risk of Wilms' tumor in his siblings and offspring. The mutational model also has important implications for cancer control. Identification of gene carriers may lead to early tumor detection and/or prevention. Identification and elimination of agents that increase germinal or somatic mutation rates may lower the incidence of hereditary or nonhereditary tumors. However, spontaneous somatic mutation rates will always maintain a certain population of patients with Wilms' tumor that must be diagnosed and treated. (87 references)

3043 ANIMAL MODEL: EMBRYO-DERIVED TERATOMAS AND TERATOCARCINOMAS IN MICE. (Eng.) Damjanov, I. (Univ. Connecticut Health Center, Farmington, CT 06032); Solter, D. *Am J Pathol* 83(1): 241-244; 1976.

Teratomas and teratocarcinomas are inducible in strains of inbred mice by transplantation of presomitic embryos into the testis or the subcapsular space between the kidney capsule and parenchyma. Teratomas, which can be derived from embryos in stages ranging from zygotes to preterm viable fetuses, are composed of somatic tissue and have a growth rate similar to the host. Usually teratocarcinomas can be obtained only from presomitic embryos and are morphologically indistinguishable from spontaneous testicular and ovarian tumors. They grow rapidly, can be transplanted and cultured, and have substantial alkaline phosphatase activity. They are composed of disorganized somatic cells and contain foci of embryonal carcinoma cells (the tumor stem cells); although they penetrate the kidney, they rarely metastasize. Teratocarcinomas, unlike teratomas, produce α-fetoprotein. Mouse teratomas and teratocarcinomas resemble human ones in the spectrum of tissue, α-fetoprotein, and alkaline phosphatase production. Because of this resemblance, these inducible mouse tumors can be used as research models for their human counterparts. (5 references)

3044 CLONAL EVOLUTION IN THE MYELOID LEUKEMIAS. (Eng.) de Grouchy, J.; Turleau, C. In: *Chromosomes and Cancer*. Edited by J. German. (New York: John Wiley & Sons): pp. 287-311; 1974.

Seven models for clonal evolution in leukemia are described and the frequency with which the various models are observed is noted. Possible mechanisms, governing laws, and the general concept of clonal evolution are discussed. The evolution of a clone is compared with the evolution of a species. (108 references)

3045 HEREDITY AND COLON CANCER. (Eng.) Lynch, H. T.; Lynch, J.; Guirgis, H. In: *Cancer Genetics*. Edited by H. T. Lynch. (Springfield, IL: Charles C. Thomas): pp. 326-353; 1976.

Hereditary syndromes associated with colon cancer are tabulated together with their mode of inheritance, anatomic location, pathology of the polyps and/or cancer, and the associated clinical features. Colon cancer in conditions that are not associated with multiple polyposis of the colon is also discussed. Research involving a retrospective and prospective series of colon cancer probands is described. Medical records were reviewed of all patients with adenocarcinoma of the colon at one hospital from 1951 to 1969 (209 probands). When possible, all living probands were interviewed. Those with doubtful histories were excluded, and 98 patients were available for the retrospective series. The prospective phase began in 1969 with a review of the medical records and interviews with each new patient with histologically verified adenocarcinoma of the colon. Each of 50 patients was age- and sex-matched to a simultaneous control patient (without cancer). All available first-degree relatives of patients and controls were interviewed, and data were compiled and analyzed as for the retrospective series. A maximum number of patients was diagnosed between the ages of 60 and 80 (mean age 64.8). Colon cancer was found in 23 first-degree relatives of the retrospective series probands and in 15 first-degree relatives of the prospective series probands. Only three cases of cancer were found in the control group. The prospective group also had an increase in breast cancer in first-degree relatives over those of the control group; multiple primary malignancies were also increased. No significant association was found between colon cancer and any other histological type except breast cancer (89 references)

3046 HODGKIN'S DISEASE--AN INFECTIOUS DISEASE? (Eng.) Vianna, N. J.; Greenwald, P.; Davies, N. J. P. In: *Hodgkin's Disease*. Edited by M. J. Lacher. (New York: John Wiley & Sons): pp. 405-415; 1976.

The epidemiological pattern of Hodgkin's disease in various communities of upstate New York, with other evidence that Hodgkin's disease is infectious, is reviewed. From 1950 through 1970, the average annual incidence of Hodgkin's disease in Albany County was 3.7/100,000, as compared with 2.5/100,000 in the other 56 upstate counties, suggesting an apparent epidemic of Hodgkin's disease in Albany County. Additional observations suggest that Hodgkin's disease may have behaved as an infectious disease in Albany: the county's average incidence was significantly higher than that of the rest of upstate N.Y. during 1953-1959 and significantly lower during 1963-1969, indicating that the epidemic period was followed by one of community immunity. An attempt was made to link the 208 cases of Hodgkin's disease diagnosed in Albany County from 1950 through 1970 either directly, or indirectly through a single contact. Of the 38 cases studied so far, 31 have been interlinked, suggesting that Hodgkin's disease is transmissible, either directly, or indirectly, through a carrier state. Apparent transmission was from young to young or old. Since the time interval between the diagnoses of most linked cases was years, it is probable that, if Hodgkin's disease is infectious, it has a mean incubation of several years. Three additional interlinked groups with the characteristics found in the Albany group were centered in schools. A fourth group with similar characteristics centered in the residents of a particular street and community. The results of a case-control study suggest that the risk of developing Hodgkin's disease in patients under 40 yr of age is 2.9 times greater if the individual has had a prior tonsillectomy. The tonsils may act as a protective barrier to the agent responsible for Hodgkin's disease. The fact that tonsillectomies are more commonly performed on children from more affluent homes may partially explain why Hodgkin's disease is disproportionately a disease of higher socioeconomic groups. Further study of the tonsillectomy hypothesis is important because, if verified, it will provide strong evidence for an infectious etiology in Hodgkin's disease. (23 references)

3047 CANCER OF THE CERVIX UTERI--AN EPIDEMIOLOGICAL SURVEY.

(Eng.) Deeley, T. J. (Velindre Hosp., Whitchurch, Cardiff, Wales). *Clin Radiol* 27: 43-51; 1976.

The epidemiology of cancer of the cervix uteri is discussed at length. Retrospective analysis has elucidated many factors that, either singly or in combination, may affect the incidence of this cancer; i.e., marriage, numerous pregnancies, frequent and early intercourse, promiscuity, smegma, and vaginal infection. The problems involved in an epidemiological study of a disease with so many factors involved, which also make it difficult to define its etiology, are discussed. The most important factor is sexual intercourse in adolescence. It is concluded that prevention requires education of the public, changes in moral standards, and improvement in socioeconomic conditions, as well as early diagnosis and treatment. (64 references)

3048 REGULATION OF GENE EXPRESSION.

(Eng.) Tomkins, G. In: *Cancer Biology. I. Induction, Regulation, Immunology and Therapy. Based on a Series of Lectures Presented at the Given Institute of Pathobiology in Aspen, Colorado, July 1974.* Edited by R. R. Pascal, F. Silva, and D. W. King. (New York: Stratton Intercontinental Medical Book Corp.): pp. 24-28; 1976.

The role of glucocorticoids and cyclic AMP in the regulation of cell replication is discussed. In cultured rat hepatoma cells at 0 C, glucocorticoid steroids penetrate the cells membranes and react with intracellular receptor proteins. The receptor:glucocorticoid complex, after undergoing further activation, develops a strong affinity for and attaches to nuclear acceptor sites containing DNA. The synthesis of 5-10 specific proteins in cultured hepatoma cells is activated by glucocorticoids. There is a good correlation between the rate of synthesis of one of these proteins, tyrosine aminotransferase, and the appearance of receptor in the nucleus. The steroids do not affect enucleated cells. Because of these observations, the site of the complex is thought to lie within the nucleus. The addition of glucocorticoids to S49 (T lymphoma cells of the mouse) blocks the growth of some cells; ultimately they die. The resistant mutant cells (arising spontaneously at $1/10^6$ cells/generation) can thus be selected. Study of the phenotypes of these resistant mutants indicates that the receptor has three domains: a nuclear DNA receptor, a steroid receptor, and an inhibitory portion. Glucocorticoids could inhibit cell growth and kill neoplastic cells (such as lymphomas) by inducing adenyl cyclase or repressing the phosphodiesterase, leading to an increase in cyclic AMP, which in turn would induce a block in G_1. S49 cyclic-AMP-resistant cells have been isolated which are missing the binding protein (cyclic nucleotide receptors) and protein kinase but which are still responsive to steroid inhibition, indicating that growth arrest by glucocorticoids is not mediated by cyclic-AMP. Transformed cells appear to have difficulty in responding to the normal signals which initiate growth arrest although the mechanism for the transition appears intact. (No references)

3049 THE GROWTH OF NORMAL AND NEOPLASTIC CELLS.

(Eng.) Stoker, M. G. P. (Imperial Cancer Res. Fund Lab., Lincoln's Inn Fields, London, England). *Clin Radiol* 27: 1-7; 1976.

The current knowledge concerning growth of normal and neoplastic cells is reviewed in order to suggest possible improvements in cancer therapy. The cell type most frequently studied is the cultured mouse fibroblast, simplest to control but difficult to identify in terms of its counterparts in vivo. Therefore, data from these studies must be interpreted with caution. The normal cell cycle and growth control factors are discussed in depth. Normal cells seem to have a period, immediately after cleavage, that is sensitive to environmental conditions. Under adverse conditions these cells will stay in the G_0 stage with no further divisions until conditions improve. Tumor and transformed cells seem to lack this ability to rest in a G_0 stage under adverse growth conditions. Instead, they continue an unbalanced growth and eventually die if conditions do not improve. Evidence indicates that tumor cells may supply some of their own control factors, which may account for their fantastic growth abilities. These facts may be applied to future therapy. That is, growth inhibitors could arrest normal cell growth at the G_0 stage (a stage in which they are highly resistant) and tumor cells could be intensively treated by chemotherapy. After treatment the normal cells could be stimulated by growth stimulants to resume normal growth. (10 references)

3050 ORAL CONTRACEPTIVES AND LIVER NODULES.

(Eng.) (No affiliation given). *Lancet* 1(7964): 843-844; 1976. (10 references)

3051 CARCINOGENIC ROLE OF VINYL CHLORIDE (Letter to Editor).

(Fre.) Pelfrene, A. F. (Univ. Nebraska Medical Center, 42nd and Dewey Ave., Omaha, NB 68105). *Nouv Presse Med* 5(15): 999-1000; 1976. (8 references)

3052 POLYNUCLEAR AROMATIC HYDROCARBONS IN RAW, POTABLE AND WASTE WATERS.

(Eng.) Harrison, R. M. (Public Health Engineering, Imperial Coll., London S.W. 7, England); Perry, R.; Wellings, R. A. *Water Res* 9(4): 331-346; 1975. (147 references)

3053 TRANSFORMATION IN VITRO BY HERPESVIRUSES--A REVIEW.

(Eng.) Pope, J. H. In: *Oncogenesis and Herpesviruses II. Part 1: Biochemistry of Viral Replication and In Vitro Transformation. Proceedings of a Symposium held in Nuremburg, Federal Republic of Germany, 14-16 October 1974.* International Agency for Res. on Cancer. (Lyon, France): pp. 367-377; 1975. (72 references)

3054 ISOLATION AND CHARACTERIZATION OF VIRUS-SPECIFIC CELL-SURFACE ANTIGENS. (Eng.) Kennel, S. J. In: *Cellular Membranes and Tumor Cell Behavior. A Collection of Papers Presented at the Twenty-Eighth Annual Symposium on Fundamental Cancer Research, 1975.* University of Texas System Cancer Center, M. D. Anderson Hospital and Tumor Institute. (Baltimore: Williams and Wilkins Co.): pp. 469-484; 1975. (31 references)

3055 SURFACE MARKERS OF HUMAN LYMPHOCYTES. (Eng.) Kunkel, H. G. (Rockefeller Univ., 1230 York Ave., New York, NY 10021). *Johns Hopkins Med J* 137(5): 216-223; 1975. (22 references)

3056 TRANSPLANTATION STUDIES, PART A. (Eng.) Reuber, M. D. In: *Mouse Hepatic Neoplasia. Proceedings of a Workshop held at the H.T.S. Management Centre, Lane End, High Wycombe (Great Britain), 12-17 May 1974.* Butler, W. H. and Newberne, P. M., eds. (New York: Elsevier Scientific Publishing Co.): pp. 144-153; 1975. (40 references)

3057 MALIGNANT TRANSFORMATIONS. (Eng.) Wohlman, A. (Armour Pharmaceutical Co., Box 511, Kankakee, IL 60901). *Perspect Biol Med* 19(1): 149; 1975. (No references)

3058 PRIOR SOCIAL LINKS AMONG LEUKEMIA AND LYMPHOMA PATIENTS. (Eng.) Vianna, N. J. (New York State Health Dept., Albany, NY). *Ann Intern Med* 84(5): 604-606; 1976. (12 references)

3059 REGULATION OF BLOOD CELL DIFFERENTIATION. (Eng.) Till, J. E. (Ontario Cancer Inst., Toronto M4X 1K9, Canada); Price, G. B.; Mak, T. W.; McCulloch, E. A. *Fed Proc* 34(13): 2279-2284; 1975. (76 references)

3060 INDUCTION OF POINT MUTATIONS BY DIFFERENT CHEMICAL MECHANISMS IN THE LIVER MICROSOMAL ASSAY. (Eng.) Herbold, B. (Institut fuer Anthropologie und Humangenetik, Univ. Heidelberg, W. Germany); Buselmaier, W. *Mutat Res* 40: 73-83; 1976.

A selection of chemical agents with different mechanisms of chemical mutability was tested by liver microsomal assay using the different bacterial tester strains: *Salmonella typhimurium* TA 1535, TA 1536, TA 1537, TA 1538, and G46. The agents are all well-known mutagens and can be divided into alkylating agents, anti-metabolites, or acridines or compounds that form radicals in the cell. The mutagens were: dimethylnitrosamine, diethylnitrosamine, methylnitronitrosoguanidine, cyclophosphamide, Captan, amethopterine, azathioprine, 6-mecaptopurine, trypaflavine, isoniazide, and hydrazine. Each substance was tested at three different doses, and the concentrations chosen depended on the sensitivity of the indicator organisms; controls received nothing. That all except amethopterine gave positive results shows that this system is very sensitive. When the different strains and mutagens were compared, a correlation was found between the diameter of the molecule and the permeability of the bacterial cell membrane: as the molecular diameter increased, the sensitivity of the G46 strain decreased and that of TA 1535 increased. This indicates that for routine screening, more consideration should be given to molecular dimensions and the effect of cell membrane permeability.

3061 THE BASE DISPLACEMENT MODEL: AN EXPLANATION FOR THE CONFORMATIONAL AND FUNCTIONAL CHANGES IN NUCLEIC ACIDS MODIFIED BY CHEMICAL CARCINOGENS. (Eng.) Grunberger, D.; Weinstein, I. B. In: *Biology of Radiation Carcinogenesis*. Edited by J. M. Yuhas; R. W. Tennant; J. D. Regan. (New York: Raven Press): pp. 175-187; 1976.

The structural and functional changes that occur in a nucleic acid when a carcinogen is bound to it were studied. The effect of N-2-acetylaminofluorene (AAF) modification of guanosine residues on base pairing and on the translation of synthetic messenger RNA was investigated. The mechanism of N-hydroxy-AAF inhibition of rat liver RNA synthesis and conformational changes in nucleic acids modified with AAF were examined. The base displacement model of chemical carcinogenesis was discussed. Results indicate that conformational changes induced in nucleic acids by AAF interfere with the normal stacking interactions between neighboring bases and with the base-pairing capacity of the modified G residues; a biologic consequence of these changes is that AAF derivatives produce frameshift mutations. (46 references)

3062 COLORIMETRIC ASSAY FOR AROMATIC AMINES. (Eng.) Rinde, E. (New York Univ. Medical Center, 550 First Ave., New York, NY 10016); Troll, W. *Anal Chem* 48(3): 542-544; 1976.

Aromatic amines can be detected in the nanomole range with the reagent Fluram, with which they form stable yellow derivatives. Fluram in glacial acetic acid reacts only with aromatic amines. The reaction is complete in ten minutes and can be performed on thin-layer (TLC) chromatograms making possible the specific measurement of aromatic amines. The yellow product can be quantitatively eluted from the TLC plates. Fluram is colorless, and the blanks are zero.

3063 DEVELOPMENT OF AN ASSAY FOR ARYL HYDROCARBON [BENZO(*a*)PYRENE] HYDROXYLASE IN HUMAN PERIPHERAL BLOOD MONOCYTES. (Eng.) Bast, R. C., Jr. (Natl. Cancer Inst., Natl. Insts. Health, Bethesda, MD 20014); Okuda, T.; Plotkin, E.; Tarone, R.; Rapp, H. J.; Gelboin, H. V. *Cancer Res* 36(6): 1967-1974; 1976.

Aryl hydrocarbon [benzo(*a*)pyrene] hydroxylase was assayed in peripheral blood monocytes from a human population. Treatment with benz(*a*)anthracene in cell culture increased aryl hydrocarbon hydroxylase activity from 6.5- to 37-fold in monocytes from each of 25 apparently healthy donors. A weak correlation was observed between the induction ratios obtained with monocytes and lymphocytes from the same donors. Reproducibilities of the monocyte and lymphocyte assays were comparable. In monocytes, the measurement of basal and induced aryl hydrocarbon hydroxylase activity did not require pretreatment with mitogens, so mitogen values were not influenced by abnormal mitogen responsiveness. Monocytes also exhibited a much wider range of induction ratios than did lymphocytes.

3064 IN VITRO MALIGNANT TRANSFORMATION OF MOUSE FIBROBLASTS BY NON-K-REGION DIHYDRODIOLS DERIVED FROM 7-METHYLBENZ(A)ANTHRACENE, 7,12-DIMETHYLBENZ(A)ANTHRACENE, AND BENZO(A)PYRENE. (Eng.) Marquardt, H. (Memorial Sloan Kettering Cancer Center, New York, NY 10021); Grover, P. L.; Sims, P. *Cancer Res* 36(6): 2059-2064; 1976.

The activities of K region and non-K region dihydrodiols derived from 7-methylbenz(a)anthracene, 7,12-dimethylbenz(a)anthracene, and benzo(a)pyrene in the in vitro induction of malignant transformation in M2 mouse fibroblasts were compared with those of the parent hydrocarbons and of the related K region epoxides. The non-K region diols, with adjacent olefinic double bonds that can be metabolized to diol epoxides, were more active than the parent hydrocarbons in inducing malignant transformation of M2 mouse fibroblasts; a fourth non-K region diol, the 9,10-dihydrodiol of benzo(a)pyrene, was less active than benzo(a)pyrene. The related K region dihydrodiols, which lack adjacent olefinic double bonds, and 6-hydroxybenzo(a)pyrene were inactive. 7,8-Dihydrobenzo(a)pyrene, a more potent carcinogen than the 9,10 isomer, induced malignant transformation, but the 9,10 isomer was inactive. Transformed cells with abnormal morphology yielded sarcomas on injection into isologous mice; treated but morphologically normal cells did not. These results support the role of diols and diol epoxides in the metabolic activation of polycyclic hydrocarbons.

3065 NONIMMUNOGENIC SARCOMAS INDUCED BY 3-METHYLCHOLANTHRENE TREATMENT OF MURINE FIBROBLASTS IN DIFFUSION CHAMBERS. (Eng.) Carbone, G. (Div. Experimental Oncology A, Istituto Nazionale per lo Studio e la Cura dei Tumori, 20133 Milan, Italy); Parmiani, G. *J Natl Cancer Inst* 55(5): 1195-1197; 1975.

A 30-µg dose of 3-methylcholanthrene (MCA) was applied for 1 wk to normal BALB/c fibroblasts in cell-impermeable diffusion chambers (DC) in the peritoneal cavities of BALB/c mice. Two groups of DC cultures (in which the carcinogen was given during the 1st and 5th wk of cultivation) were compared for the frequency of malignant transformation and for the immunogenicity of the resulting neoplasms. The cells from each DC were transplanted sc into immunodepressed semisyngeneic mice for assay of their tumorigenicity. Although tumor yield was similar in the two groups (25 and 22%), there was clear difference in immunogenicity; 10 of 16 sarcomas from fibroblasts treated during the 1st wk of culture were nonimmunogenic, whereas 8 of 9 tumors from the older cultures were immunogenic. A kinetic study of normal fibroblasts in DC revealed that cells proliferated rapidly, with a peak at the 4th day after seeding, then grew progressively more slowly and ceased to replicate between 14 and 28 days of culture. There was a notable difference at the moment of the MCA application in the growth phase of the target cell population of the 1st as compared with the 5th week of culture.

3066 INTERACTION OF THE CARCINOGEN 3,3-DIMETHYL-1-PHENYLTRIAZENE WITH NUCLEIC ACIDS OF VARIOUS RAT TISSUES AND THE EFFECT OF A PROTEIN-FREE DIET. (Eng.) Kleihues, P. (Max-Planck-Institut fuer Hirnforschung, Cologne, W. Germany); Kolar, G. F.; Margison, G. P. *Cancer Res* 36(7): 2189-2193; 1976.

The methylation by 3,3-(^{14}C)dimethyl-1-phenyltriazene, (^{14}C)DMPT, of nucleic acids in various rat tissues was investigated. Following a single sc injection of (^{14}C)DMPT (50 mg/kg), approximately 40% of the radioactivity was subsequently exhaled as $^{14}CO_2$. Expiration of $^{14}CO_2$, metabolic ^{14}C labeling of liver proteins, and formation of 7-(^{14}C)methylguanine, the major reaction product with nucleic acid bases, were completed within about 15 hr. Minor alkylation products detectable in DNA were O^6-methylguanine and 3-methyladenine. In cytoplasmic RNA, 1-methyladenosine, 3-methylcytidine, O^6-methylguanosine and 7-methylguanosine were present. Concentrations of 7-methylguanine were highest in nucleic acids of kidney and liver. Among the other organs investigated (brain, lung, spleen, and small intestine), 7-methylguanine levels showed little variation but were four to seven times lower than those in liver and kidney. Feeding of a protein-free diet prior to (^{14}C)DMPT administration reduced the formation of 7-methylguanine in liver and kidney RNA, whereas in the remaining organs the extent of methylation was markedly increased. Methylation at specific sites in nucleic acids for the initiation of malignant transformation and the possible role of 3-methyl-1-phenyltriazene as the systemically distributed proximate carcinogen of 3,3-dimethyl-1-phenyltriazene are also discussed. (29 references)

3067 FORMATION OF TRIHALOMETHANES BY CHLORINATION OF SURFACE WATER. (Eng.) Bunn, W. W. (U.S. Environmental Protection Agency, Region VII, Kansas City, KS 66115); Haas, B. B.; Deane, E. R.; Kleopfer, R. D. *Environ Lett* 10(3): 205-213; 1975.

The occurrence of the previously undetected trihalogenated methane, dichloroiodomethane, in tap water from the Meramec and the Missouri Rivers is reported. A gas chromatographic/mass spectrometric analysis of resin extracts was performed. Experimental evidence indicates that all 10 possible trihalogenated methanes (Cl, Br, and I, but not F) can be formed by chlorination of surface water. The addition of KF or KCl to subsequently chlorinated Missouri River water had little effect on the production of trihalogenated methanes. On the other hand, KBr or KI caused substantial reduction in chloroform concentrations with the formation of bromodichloromethane, dibromochloromethane, and bromoform or dichloroiodomethane, chlorodiiodomethane, and iodoform, respectively. Oxidation of bromide and iodide to hypobromite and hypoiodite plus a subsequent ''haloform'' type reaction is a possible explanation for the formation of these compounds.

3068 FOCAL LOSS OF ALKALINE PHOSPHATASE AND INCREASE OF PROLIFERATION IN PRENEOPLASTIC AREAS OF THE RAT UROTHELIUM AFTER ADMINISTRATION OF N-BUTYL-N-(4-HYDROXYBUTYL)-NITROSAMINE AND N-[4-(5-NITRO-2-FURYL)-2-THIAZOLYL] FORMAMIDE. (Eng.) Kunze, E. (Inst. Pathology, Univ. Goettingen, D-3400 Goettingen, GoBlerstr. 10, W. Germany); Schauer, A.; Kruesmann, G. *Z Krebsforsch* 84(2): 143-160; 1975.

The development of tumors of the urinary bladder was studied in 59 male and female Sprague-Dawley and Wistar rats with combined enzyme-histochemical and autoradiographic methods after application (po) of n-butyl-n-(4-hydroxybutyl)-nitrosamine (BBN) and n-[4-(5-nitro-2-furyl)-2-thiazolyl] formamide (FANFT). As the first carcinogenic lesion detectable by light microscopy, a focal, sharply defined irreversible loss of alkaline phosphatase activity was consistently demonstrated in the urothelium. NADH-diaphorase activity was markedly reduced in identical regions. The enzyme-deficient areas were considered as preneoplastic, because papillomas and carcinomas developed from them through different stages of hyperplasia. These also were characterized by total loss of alkaline phosphatase activity and attenuation of the NADH-diaphorase in all parts or circumscribed areas. Autoradiographically ^{3}H-thymidine-labeling index revealed a 43.2-fold (BBN) and 22.6-fold (FANFT) increase in the enzyme-deficient areas. After 54 hr of continuous labeling there was a mean ^{3}H-thymidine-labeling index of 54.9% in the enzyme-negative regions. The physiological mode of regeneration was no longer maintained in the areas of enzyme deficiency, as there was an increased proliferation of suprabasal cells. Areas of papillomas that showed a marked attenuation of NADH-diaphorase had a ^{3}H-thymidine-labeling index greater than the surrounding areas with preserved enzyme activity.

3069 REFERENCE RAMAN SPECTRA OF DDT AND FIVE STRUCTURALLY RELATED PESTICIDES AND OF FIVE PESTICIDES CONTAINING THE NORBORNENE GROUP. (Eng.) Nicholas, M. L. (Dept. Chemistry, Coll. Wooster, Wooster, OH 44691); Powell, D. L.; Williams, T. R.; Bromund, R. H. *J Assoc Off Anal Chem* 59(1): 197-208; 1976.

The Raman spectra of two groups of pure halogenated pesticides were recorded to provide accurate spectra as an aid to identification of the compounds. The first group included DDT, TDE, Perthane, methoxychlor, dicofol, and tetradifon, and the second, the norbornene pesticides: heptachlor, chlordane, dieldrin, endrin, and endosulfan. Despite similarities for all spectra in the 3,000 cm^{-1} region, in strong peaks below 600 cm^{-1}, in a strong peak near 1,600 cm^{-1}, and in activity between 600 and 1,600 cm^{-1}, diagnostic peaks could be discerned for many of the pesticides. Dicofol is the only compound that exhibits a doublet at 3,546 and 3,533 cm^{-1}. DDT vs TDE is distinguished by a strong peak at 394 cm^{-1} (DDT) or a medium peak at 243 cm^{-1} (TDE). Similarly, the geometric isomers, dieldrin and endrin, are distinguished by peaks at 254 cm^{-1} (dieldrin) and 224 cm^{-1} (endrin). Purification of <1 mg of sample by techniques such as thin-layer chromatography is necessary before identification of the pesticides by Raman spectroscopy.

3070 NEOPLASTIC TRANSFORMATION OF FETAL RAT BRAIN CELLS IN CULTURE AFTER EXPOSURE TO ETHYLNITROSOUREA IN VIVO. (Eng.) Laerum, O. D. (Dept. Pathology, Univ. Bergen, 5000 Bergen, Norway); Rajewsky, M. F. *J Natl Cancer Inst* 55(5): 1177-1187; 1975.

The single, transplacental pulse of N-ethyl-N-nitrosourea (ENU; 75 μg/g body wt) to the fetal (18th day of gestation) BD IX rat led to death with malignant tumors of the CNS and peripheral nervous system after a median time of 195 days. Dissociated brain cells transferred to long-term cell culture 20-90 hr after the ENU pulse became tumorigenic after 200 days (as assayed by reimplantation into baby BD IX rats). This was preceded by a characteristic sequence of phenotypic alterations (termed ''stages I-IV''). During early primary culture (stage I), both ENU and control cultures exhibited stationary glia-like cells on a growing layer of epithelioid and few fibroblast-like cells. Stage II (10th-40th day) was characterized by a constant proportion of glia-like cells in the ENU cultures and by their disappearance in the controls. During stage III (40th-100th day), slowly proliferating glia-like cells in the ENU cultures formed ''piled-up'' foci. They could then be removed from the underlying cell layer and cultured separately. Transition to stage IV (100th-200th day) was marked by proliferation of morphologically altered cells, which subsequently acquired the capacity to form colonies in semisolid agar and became tumorigenic (stage V).

3071 EXPERIMENTAL INDUCTION OF CHORIOEPITHELIOMA IN RATS. (Eng.) Hosokawa, T. (Jikei Univ. Sch. Medicine); Ito, H.; Sekine, T.; Komuro, N.; Tanaka, T. *Jikeikai Med J* 22(4): 237-242; 1975.

In an attempt to induce chorioepithelioma experimentally, the placentas of 185 pregnant Wistar-Imamichi rats were injected with dimethylbenzanthracene (0.1 ml of 15% rat emulsion) or with a 30% acetone emulsion of 4-nitroquinoline oxide (0.1 ml) on the 12th to 15th day of gestation. 17α-Hydroxyprogesterone capronate was then injected im once weekly for 20 wk. Of the 92 animals that survived the experiment, two treated with 4-nitroquinoline oxide developed chorioepitheliomas, and eight treated with dimethylbenzanthracene developed the tumor. The tumor consisted of cells resembling small basophilic cells of the basal zone in the normal placenta. The activity of 3-β-ol dehydrogenase was identified in the cytoplasm of the tumor cells. The results suggest that the induced tumor is derived from the basophilic cells of the placenta, and the mutational change of these cells may promote the development of gestational chorioepithelioma.

3072 N-NITROSAMINE FORMATION FROM ATRAZINE. (Eng.) Wolfe, N. L. (Southeast Environmental Res. Lab., U.S. Environmental Protection Agency, Athens, GA 30601); Zepp, R. G.; Gordon, J. A.; Fincher, R. C. *Bull Environ Contam Toxicol* 15(3): 342-347; 1976.

The N-nitrosamine of the pesticide atrazine was studied because (a) N-nitrosamines are potent carcinogens in humans, (b) atrazine is the most widely used pesticide in the U.S., and (c) it has been found in the drinking water in New Orleans and the Midwest. Synthetic reaction conditions yielded 2-chloro-4-(N-nitroso-N-ethylamino)-6-isopropylamino-s-triazene (NNA). Two other expected products were not isolated. The compound was characterized and a structure assigned. Maximum formation occurred at pH 1.8, and the reaction mechanism seemed to be third-order. NNA kinetics were compared to those of N-nitrosamines formed from other pesticides. NNA was extremely stable in any water solutions above pH 4; however, it was rapidly decomposed by light. This instability in light may prevent the buildup of NNA in environmental samples. Therefore, the most likely location for formation of NNA is in the soil. Studies have demonstrated that atrazine and probably NNA (since it is more water-soluble than atrazine) are widespread in groundwaters of the Midwest as a result of leaching from the soil.

3073 INFLUENCE OF SEX HORMONES ON KIDNEY TUMORS INDUCED IN RATS WITH N-BUTYLNITROSOUREA. (Eng.) Takizawa, S. (Res. Inst. Nuclear Medicine and Biology, Hiroshima Univ., Kasumi 1-2-3, Hiroshima 734, Japan). *Gann* 67(1): 33-40; 1976.

The influence of sex hormones on kidney tumorigenesis induced by N-butylnitrosourea (BNU) was studied. Thirty-nine kidney tumors were induced by a continuous po administration of BNU (5 mg/15 ml drinking water) in 33 out of 204 4-wk-old W/Fu strain rats and 41 (W/Fu x ACI/N)F_1 rats. No spontaneous renal tumors were observed among 66 males and 109 females of W/Fu rats that survived beyond the age of 19 mo. Histologically, renal cell and mesenchymal types were commonly observed, 24 cases belonging to the former and 11 cases to the latter. Two cases of nephroblastoma were also encountered. There was no sex difference in renal tumorigenesis with BNU as a whole. Castration in both sexes was apparently inhibitory for kidney tumor development. Estrogenization of ovariectomized or orchidectomized rats either by syngeneic ovary graft or by repeated injections of estradiol benzoate (0.01 mg, twice weekly for 7 mo) enhanced tumor induction with BNU. Progesterone (0.4 ng, twice weekly, for 7 mo) was not effective in restoring the tumor incidence in castrated rats. Distribution of histological types of tumors thus induced differed among the hosts with different hormonal conditions; in males the majority was the renal cell type, whereas almost all mesenchymal tumor and nephroblastoma cases were found in intact females or estrogenized rats. BNU induced a variety of tumors in several organs, including the cerebral hemisphere, peripheral nerves, mammary glands, hematopoietic system, and digestive tracts. However, such concurrence did not affect renal tumor development in the present study.

3074 ON THE SIGNIFICANCE OF SEX HORMONES IN PRODUCING EXPERIMENTAL PROSTATE TUMOR IN THE RAT. (Eng.) Higuchi, M. (Kurume Univ. School Med., Kurume, 830, Japan). *Kurume Med J* 22(3): 113-134; 1975.

Modulation of the chemical induction of prostate tumors by hormonal factors was investigated. The incidences of prostate tumors in young male Donryu rats whose prostate glands were directly injected with 0.1 ml of a 1% solution of 20-methylcholanthrene (20-MC) or 4-nitroquinoline-N-oxide (4-NQO) in olive oil were 44.4% and 40%, respectively. When a 2.5 mg of testosterone propinate were injected sc each day throughout the period of observation, the incidences were 33.3% and 36.3% after 20-MC and 4-NQO, respectively. In castrated rats the incidences were 60.0% and 66.6%, respectively, and in castrated rats injected with 0.5 mg/day hexestrol they were 66.6% for both 20-MC and 4-NQO treated groups. The insertion in the sc region of 0.1-mg crystals of 20-MC or 4-NQO wrapped in 2 mm^3 of prostate parenchymal tissue derived from young male rats given 2.5 mg/d x4 of testosterone propinate sc resulted in the following respective incidences: 27.2% and 11.2% in untreated male rats, 25.0% and 9.1% following administration of testosterone propinate, 33.3% and 37.5% in castrated animals, 50.0% and 33.3% in castrated animals following hexestrol, and 7.1% and 9.1% in untreated female rats. It is thus apparent that castration and castration with estrogen increased the rate of tumor formation, and that androgen administration tended to reduce it. The tumors formed by both protocols were adenomas, squamous cell carcinomas, and sarcomas: no adenocarcinomas were observed. Further studies were made of the properties of the tumors formed after sc transplantation to other rats of the same strain: 4% of adenomas transplanted to female animals changed to adenocarcinomas. (35 references)

3075 HYDROXYLATION OF THE XENOBIOTICS AND STEROID HORMONES IN THE ENDOPLASMIC RETICULUM OF THE RAT LIVER. (Rus.) Sergeev, P. V. (Second Moscow N. I. Pirogov Medical Inst., Moscow, USSR); Khalilov, E. M.; Archakov, A. I.; Seifulla, R. D.; Devichenskii, V. M.; Karuzina, I. I. *Biull Eksp Biol Med* 81(3): 299-301; 1976.

A new polarographic technique for measuring the oxidation rate of steroid hormones and xenobiotics is discussed. Protein content was determined in the liver microsomes of male random-bred albino rats. Oxygen consumption was measured by an LP-60 polarograph with a fixed exposed platinum electrode. Hydroxylation rate was calculated as the difference between oxygen consumption rates in the presence of substrates (ethylmorphine, dimethylalanine, amidopyrine, and aniline) and in their absence. The oxidation rate was maximum for ethylmorphine and minimum for aniline. Hydroxylation rate in the group of steroid hormones (androgens, estrogens, corticosteroids, gestagens, and cholesterol) was found to depend on the polarity of the steroid molecule. (8 references)

3076 CYTOKININS IN *VINCA ROSEA* L. CROWN GALL TUMOR TISSUE AS INFLUENCED BY COMPOUNDS CONTAINING REDUCED NITROGEN. (Eng.) Peterson, J. B. (Dept. Plant Sciences, Indiana Univ., Bloomington, IN 47401); Miller, C. O. *Plant Physiol* 57(3): 393-399; 1976.

3077 ON THE ISOLATION OF TI-PLASMID FROM AGROBACTERIUM TUMEFACIENS. (Eng.) Ledeboer, A. M. (Dept. Biochemistry, State Univ. Leiden, Wassenaarseweg 64, Leiden, The Netherlands); Krol, A. J. M.; Dons, J. J. M.; Spier, F.; Schilperoort, R. A.; Zaenen, I.; Van Larebeke, N.; Schell, J. *Nucleic Acids Res* 3(2): 449-464; 1976.

3078 EVIDENCE FOR DIVERSE TYPES OF LARGE PLASMIDS IN TUMOR-PRODUCING STRAINS OF *AGROBACTERIUM*. (Eng.) Currier, T. C. (Dept. Crop and Soil Sciences, Michigan State Univ., East Lansing, MI 48824); Nester, E. W. *J Bacteriol* 126(1): 157-165; 1976.

3079 CYTOKININ-ACTIVE NUCLEOSIDES IN *AGROBACTERIUM TUMEFACIENS* TRANSFER RNA (Proceedings Abstract). (Eng.) Morris, R. O. (Agricultural Chemistry, Oregon State Univ., Corvallis, OR 97331); Chapman, R. W. *Fed Proc* 35(7): 1469; 1976.

3080 ABSORPTION AND DISTRIBUTION OF STERIGMATOCYSTIN IN THE RAT (Proceedings Abstract). (Eng.) Moore, M. (College Pharmacology, Univ. Texas, Austin, TX 72712); Scholler, J.; Smith, R. V.; Ziegler, D. *Fed Proc* 35(3): 327; 1976.

3081 MICROSOMAL N-HYDROXYLATION OF THE CARCINOGEN, N-2-FLUORENYLACETAMIDE (2-FAA) (Proceedings Abstract). (Eng.) Gutmann, H. R. (VA Hosp. and Dept. Biochemistry, Univ. Minnesota, Minneapolis, MN 55417); Malejka-Giganti, D.; Bell, P. *Fed Proc* 35(7): 1421; 1976.

3082 STRUCTURAL REQUIREMENTS FOR MUTAGENESIS. ACYL ANALOGS OF 2-ACETYLAMINOFLUORENE (Proceedings Abstract). (Eng.) Nelson, W. L. (Section on Molecular Toxicology, Developmental Pharmacology Branch, NICHD, Bethesda, MD 20014); Thorgeirsson, S. S. *Fed Proc* 35(7): 1707; 1976.

3083 EFFECTS OF TALLOW AND CORN OIL ON THE MUTAGENICITY AND CARCINOGENICITY OF 2-ACETYLAMINOFLUORENE (2-AAF) IN RATS (Proceedings Abstract). (Eng.) Castro, C. E. (Texas Tech. Univ. Complex, Lubbock, TX 79409); Felkner, I. C.; Yang, S. P.; Sproat, H. F. *Fed Proc* 35(3): 519; 1976.

3084 THE RELATIONSHIP OF 2-ACETAMIDOFLUORENE MUTAGENICITY IN PLATE TESTS WITH ITS IN VIVO LIVER CELL COMPONENT DISTRIBUTION AND ITS CARCINOGENIC POTENTIAL. (Eng.) McGregor, D. (Section Mutagen, Teratogen and Carcinogen Testing, Inveresk Res. International, Inveresk Gate, Musselburgh, EH21 7UB, Midlothian, Great Britain). *Mutat Res* 30(3): 305-315; 1975.

3085 MODIFICATION OF DNA WITH N-2-ACETYLAMINOFLUORENE (AAF): EFFECT ON CHROMATIN STRUCTURE AND TEMPLATE ACTIVITY (Proceedings Abstract). (Eng.) Yamasaki, H. (Columbia Univ., Coll. Physicians and Surgeons, New York, NY 10032); Leffler, S.; Poupko, J.; Weinstein, I. B. *Fed Proc* 35(7): 1518; 1976.

3086 ACTIVATION OF THE *O*-GLUCURONIDE OF THE CARCINOGEN *N*-HYDROXY-*N*-2-FLUORENYLACETAMIDE BY ENZYMATIC DEACETYLATION *IN VITRO*: FORMATION OF FLUORENYLAMINE-tRNA ADDUCTS. (Eng.) Cardona, R. A. (Uniroyal Chemical, Division Uniroyal, Inc., Agricultural Chemicals Res., Naugatuck, CT 06770); King, C. M. *Biochem Pharmacol* 25(9): 1051-1056; 1976.

3087 INDUCTION OF POSTREPLICATION REPAIR OF DNA FROM CHINESE HAMSTER CELLS TREATED WITH N-ACETOXY-ACETYLAMINOFLUORENE (Proceedings Abstract). (Eng.) D'Ambrosio, S. M. (Biology Dept. Brookhaven Natl. Lab., Upton, NY 11973). *Fed Proc* 35(7): 1490; 1976.

3088 ALTERATIONS IN THE TRANSCRIPTIONAL CAPACITY OF HEPATIC RNA POLYMERASE BY N-HYDROXY-2-ACETYLAMINOFLUORENE (N-OH-AAF) (Proceedings Abstract). (Eng.) Glazer, R. I. (Emory Univ., Atlanta, GA 30322). *Proc Am Assoc Cancer Res* 17: 58; 1976.

3089 GAP JUNCTIONS IN CHEMICAL CARCINOGEN (FANFT)-INDUCED TRANSITIONAL CELL CARCINOMAS IN RAT URINARY BLADDER (Proceedings Abstract). (Eng.) Pauli, B. U. (Depts. Pathology, Rush Medical Coll., Chicago, IL 60612); Weinstein, R. S.; Alroy, J.; Cohen, S. *Fed Proc* 35(3): 476; 1976.

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See also:

* (Rev): 3001, 3002, 3003, 3004, 3005, 3006, 3007, 3008, 3009, 3011, 3012, 3013, 3014, 3015, 3016, 3017, 3018, 3019, 3020, 3021, 3022, 3037, 3050, 3051, 3052, 3056
* (Phys): 3145
* (Viral): 3200, 3207, 3264, 3265, 3271, 3273
* (Immun): 3344, 3352, 3354, 3359, 3384, 3391
* (Path): 3394, 3400, 3408, 3411, 3414
* (Epid-Biom): 3431, 3432, 3434, 3435, 3437, 3438, 3446, 3447, 3449

3143 MOUSE SPLEEN LYMPHOCYTE BACTERIAL AND PEROXIDASE ACTIVITIES: ENHANCEMENT BY WHOLE BODY X-IRRADIATION (39349). (Eng.) Paul, B. B. (St. Margaret's Hosp., Boston, MA 02125); Poskitt, P. K. F.; Selvaraj, R. J.; Zgliczynski, J. M.; Sbarra, A. J. *Proc Soc Exp Biol Med* 152(2): 151-155; 1976.

Peroxidase and bacterial activities in the 20,000 g pellet of homogenates of CD mouse spleen are reported. Total body irradiation (400 R) 1 day prior to preparation of the homogenate resulted in an approximately fivefold increase in peroxidase activity, and the bactericidal activity against *Escherichia coli* was approximately doubled in preparations from irradiated mice. The rate of glucose oxidation, as measured by the formation of $^{14}CO_2$ from ^{14}C-labeled glucose, was studied in these nonphagocytic spleen cells. Addition of polystyrene latex particles to these cells from normal or x-irradiated animals increased glucose oxidation three to four times. With increasing doses of radiation (100-800 R, 1 day prior to measurement) there was increasing depression of spleen wt, total spleen cells, and total peripheral WBC. The proportion of peripheral and spleen lymphocytes decreased, while that of segmented cells increased. Ten to 15 days after irradiation, all parameters had returned to normal. Stimulation of glucose oxidation by particle addition may be the result of interaction of the particles with the cell surface. The increased bactericidal effect due to x-irradiation may result from the increased peroxidase activity. Surface action rather than phagocytosis may be involved in the bactericidal process. Differences between lymphocyte peroxidase and myeloperoxidase are discussed. (16 references)

3144 MALIGNANT TUMORS IN PEOPLE EXPOSED AS CHILDREN TO THE ATOMIC BOMB IN NAGASAKI. (Jap.) Ikeda, T. (Nagasaki Univ. Sch. Medicine, Nagasaki, Japan); Matsuo, T.; Mori, Y.; Nonaka, M.; Oribe, T.; Yun, K.; Fujiwara, H. *Hiroshima Igaku* 29(3): 264-269; 1976.

A total of 88 cases of malignant tumors were investigated in patients who had been exposed to the atomic bomb in Nagasaki during their childhood (ages 0-9 yr). As controls, 286 patients with malignant tumors who had not been exposed were also studied. The investigations included determination of sex, age when exposed, amount of radiation, distance from atomic blast center, age when the malignant tumor was diagnosed, location of the tumor, and histopathological classification of the tumor. Due to the insufficient number of cases, conclusions could not be drawn with any certainty. However, the evidence suggested the following: (a) most of the tumors among the patients who had been exposed to radiation were diagnosed as malignant lymphoma, thyroid cancer, cephaloma, sarcoma, and ovarian cancer. (b) Females suffered more gastric cancer and malignant lymphoma than males, who showed a higher percentage of sarcoma and cephaloma. (c) The following cancers were found less frequently among the group exposed to radiation than the control group: cervical cancer, colon cancer, breast cancer, pharyngoesophageal cancer, embryoma, and chorioma. (d) Three types of cancer, thyroid cancer, cephaloma, and gastric cancer, appear to be related to exposure of the patients to radiation during childhood. This relationship was determined by examining the distance from the blast center to the patient, the latent period from exposure to diagnosis, and the histopathological determination of the malignant tumor. (9 references)

3145 INFLUENCE OF UV IRRADIATION, 4-NITROQUINOLINE-1-OXIDE, METHYLMETHANESULFONATE, AND BLEOMYCIN ON THE ACTIVITY OF AN ALKALINE DEOXYRIBONUCLEASE FROM HUMAN LYMPHOCYTES. (Eng.) Zoellner, E. J. (Physiologisch-Chemisches Institut der Johannes Gutenberg-Universitaet, 65 Mainz, W. Germany); Weinblum, D.; Obermeier, J. *Exp Cell Res* 99(1): 185-189; 1976.

The in situ assay of deoxyribonucleases in DNA-containing polyacrylamide gels was used to determine the influence of UV irradiation. 4-nitro-quinoline-1-oxide, methylmethanesulfonate, and bleomycin on the activity of an alkaline deoxyribonuclease from human lymphocytes. The activity of this endonuclease, specific for denatured or UV-irradiated DNA, increased in UV-irradiated cells and in cells incubated with 4-nitroquinoline-1-oxide and bleomycin. The increment was not altered by hydroxyurea (1 mM), which inhibited DNA synthesis or caffeine (0.15 mg/ml), which prevents recombinational repair. The nuclease probably is involved in repair replication of mispaired DNA regions.

3146 PATHWAYS IN MURINE RADIATION LEUKEMOGENESIS-COLEUKEMOGENESIS. (Eng.) Haran-Ghera, N. In: *Biology of Radiation Carcinogenesis*. Edited by J. M. Yuhas; R. W. Tennant; J. D. Regan. (New York: Raven Press): pp. 245-260; 1976.

An experimental system involving the induction of lymphatic leukemia in C57BL/6 mice by exposing them to fractionated whole-body irradiation or by inoculating them with radiation leukemia virus was investigated. Within 4 wk after the radiation treatment was terminated, preleukemic cells were found in the bone marrow of irradiated mice, although they were clinically normal for several months before overt disease was observed. Methods for the prevention or reduction of radiation leukemogenesis did not affect the degree of leukemogenic agent 'release' in irradiated bone marrow nor did they decrease the level of neoplastic transformation among the irradiated bone marrow cells. Although older mice are less susceptible to overt leukemia, the increased age (9 mo) did not affect the susceptibility of lymphoid cells to neoplastic transformation. Overt leukemia development could be related to the ultimate capacity of preleukemic cells to escape immune surveillance. Another possibility is that within the long latent period, the early preleukemic cells could acquire new properties that would allow uncontrolled proliferation. Fractionated radiation increased the incidence of thymocytes bearing high levels of H-2 alloantigens. The transient availability of this high H-2 thymus subpopulation might be a prerequisite for the development of overt leukemia. The initial direct virus-thymus cell interaction led to high leukemia induction potency without the need for further host treatment, whereas virus-bone marrow interaction would yield a low leukemia incidence unless a change in the virus-lymphoid cell relationship was introduced. Radiation might act as a co-leukemogenic agent because it can change the virus/T and B cell interactions. (25 references)

3147 PLASMA ALKALINE PHOSPHATASE IN MICE WITH EXPERIMENTALLY-INDUCED OSTEOSARCOMAS.

(Eng.) Bailey, J. M. (Dept. Microbiology, Montana State Univ., Bozeman, MT 59715); Hill, W. D.; Fiscus, A. G.; Reilly, C. A.; Finkel, M. P. *Lab Anim Sci* 26(1): 66-69; 1976.

Plasma alkaline phosphatase levels were measured in CF1/Anl mice before and during growth of osteosarcomas induced by (a) injection of FBJ viral extract, (b) exposure to whole-body roentgenograms, or (c) injection of ^{90}Sr. Measurements were made at various times during tumor growth, and at no time was there a statistically significant difference in enzyme level associated with FBJ-osteosarcoma or exposure to x-ray. However, the plasma alkaline phosphatase levels were useful in predicting osteosarcoma induced by ^{90}Sr. That is, in 81% of the treated mice (^{90}Sr), elevated levels of the enzyme were found before or at the same time as positive identification of bone cancer. Various explanations are presented to account for these results. This enzymatic technique for determining bone cancer in mice is much easier and less expensive than the x-ray method previously used. The benefit to investigators is obvious, although the enzyme technique gives no information on tumor location.

3148 CHARACTERIZATION OF NATURAL ANTIBODIES IN MICE TO ENDOGENOUS LEUKEMIA VIRUS. (Eng.)

Ihle, J. N.; Lee, J. C.; Hanna, M. C., Jr. In: *Biology of Radiation Carcinogenesis*. Edited by J. M. Yuhas; R. W. Tennant; J. D. Regan. (New York: Raven Press): pp. 261-273; 1976.

Techniques and findings are reviewed concerning the autogenous immune response in relation to the immunosuppressive effects of radiation in the induction of thymic lymphomas in mice. A sensitive radioimmune precipitation assay that uses ^{3}H-leucine-labeled AKR leukemia virus was selected to establish the presence of natural antibody to the virus and to assess the humoral response quantitatively. When age-matched sera from several strains of mice were assayed, striking variations in the titers that appeared to correlate to the incidence of spontaneous leukemias were apparent. Strains such as BALB/c, C57BL/6, and C3H have intermediate titers and a 10%-20% incidence of spontaneous leukemias. Strains with a high incidence, such as RF and AKR, generally have low and variable titers against the virus. A group of strains such as NIH have a low leukemia incidence and low levels of antibody. The data suggested that susceptible strains have endogenous ecotropic virus and intermediate or low titers of antibody. Further experiments showed a high degree of specificity of the autogenous immune response in terms of antigenic recognition and antibody type. It was observed that only the 19S immunoglobulins react with gp71; this is significant in terms of radiation-induced pathogenesis. (22 references)

3149 GAMMA-RAY EXCISION REPAIR IN NORMAL AND DISEASED HUMAN CELLS. (Eng.)

Cerutti, P. A.; Remsen, J. F. In: *Biology of Radiation Carcinogenesis*. Edited by J. M. Yuhas, R. W. Tennant, J. D. Regan. (New York: Raven Press): pp. 93-101; 1976.

Radiation products of the 5,6-dihydroxydihydrothymine type (t') are efficiently removed from DNA during postirradiation incubation of bacterial and mammalian cells. The authors describe the t'-excision system contained in normal human cells, in human carcinoma HeLa S-3 cells, and in skin fibroblasts from xeroderma pigmentosum (XP) and Fanconi's anemia (FA) patients. The properties of the t'-excision repair system of human cells were studied with the use of whole cell sonicates and purified nuclear preparations of HeLa S-3 cells and γ-irradiated bacteriophage DNA as exogenous substrate. The reaction was maximum (about 50% of t' removed within 40 min of incubation at 37 C) at the lowest initial t' concentration of 0.3 x 10^{-6} μmole t'/sample. Unspecific degradation of the exogenous DNA substrate was only 2%-3% within 60 min incubation. The t' excision process is therefore highly selective. The t' capacity of preparations from human skin fibroblasts was similar to that described for HeLa cells. A depression or complete absence of unscheduled DNA synthesis was found in most XP cells after UV exposure but not after exposure to ionizing radiation. It was concluded that nuclear preparations from the Fanconi cell lines possess a decreased capacity for the excision of γ-ray-damaged thymine. (45 references)

3150 α-S-CYSTEINYLTHYMIDINE: A MODEL FOR PROTEIN-NUCLEIC ACID CROSS-LINKING. (Eng.)

Berman, H. M. (Inst. for Cancer Res., Fox Chase Cancer Center, Philadelphia, PA 19111); Zacharias, D. E.; Carrell, H. L.; Varghese, A. J. *Biochemistry* 15(3): 463-467; 1976.

Crystals of alpha-S-cysteinylthymine, $C_8H_{12}ClN_3O_4S$, formula wt 281.72, were found to be orthorhombic, space group $P2_12_12_1$, with a = 9.499 (1), b = 24.072 (4), and c = 5.012 (1) A, V = 1146.1 (2) A^3, and Z = 4. Three-dimensional data were collected on a Syntex P1 automated diffractometer with monochromated Cu K alpha radiation using the variable theta-2theta scan technique in the sin theta/lambda range 0-0.603 A^{-1}. Intensities were measured for 1402 reflections. The raw data were corrected for intensity loss by means of a linear plot derived from the change in intensity of measured standard reflections as a function of exposed time. Values of sigma(F) were derived from counting statistics and measured instrumental uncertainties. The data were reduced to a unique set of 1277 independent reflections (80% of the 1587 theoretically accessible) and converted to structure amplitudes. A Wilson plot of the data produced a scale factor to place the data on an absolute scale. No x-ray absorption correction was applied. The structure was solved by direct methods by means of the program MULTAN. The first E map revealed all of the non-hydrogen atoms. After several cycles of isotropic and anisotropic least-squares refinement, a difference map was calculated which revealed the positions of the hydrogen atoms. Further refinement with anisotropic temperature factors (isotropic temperature factors for hydrogen) converged at residual, R, of 0.043, wR = 0.053. The weighting scheme was derived from an analysis of variance. For $F < 80$, sigma = 4.5; for $F > 80$, sigma = 4.5 + 0.35(F-80). From the structure, a three-dimensional model for the radiation-induced interaction of thymine residues and cysteine residues could be postulated.

3151 EXCISION OF PYRIMIDINE DIMERS FROM EPIDERMAL DNA AND NONSEMICONSERVATIVE EPIDERMAL DNA SYNTHESIS FOLLOWING ULTRAVIOLET IRRADIATION OF MOUSE SKIN. (Eng.) Bowden, G. T. (Univ. Wisconsin Medical Sch., Madison, WI 53706); Trosko, J. E.; Shapas, B. G.; Boutwell, R. K. *Cancer Res* 35(12): 3599-3607; 1975.

The pyrimidine dimer production and excision in epidermal DNA were studied at five different dose levels of UV light in the skin of intact mice. Dimer production increased with dose up to 50,400 ergs/mm^2. About 30% of the thymine-containing dimers were excised by 24 hr after irradiation at three lower dose levels of UV light. Nonsemiconservative DNA replication in UV-irradiated mouse skin was shown to continue for 18 hr. The rate of nonsemiconservative replication decreased with time. The initial rates of nonsemiconservative replication increased with UV light dose level up to 4,200 ergs/mm^2, after which the initial rates were decreased. Semiconservative DNA synthesis was shown to be inhibited by hydroxyurea; however, hydroxyurea had no effect on UV light-induced nonsemiconservative DNA replication. The pyrimidine dimer excision and nonsemiconservative DNA replication suggested that, in the intact mouse, the cells of the epidermis were capable of DNA excision repair after UV irradiation of the mouse skin.

3152 DATA ON THE ACTION SPECTRUM FOR ULTRAVIOLET CARCINOGENESIS. (Eng.) Freeman, R. G. (Univ. Texas Southwestern Medical Sch., Dallas, TX 75235). *J Natl Cancer Inst* 55(5): 1119-1122; 1975.

See also:

* (Rev): 3001, 3002, 3021, 3022
* (Viral): 3158, 3161, 3264
* (Path): 3414
* (Epid-Biom): 3446

3153 TRANSFORMATION-DEFECTIVE MUTANTS OF AVIAN SARCOMA VIRUSES: THE GENETIC RELATIONSHIP BETWEEN CONDITIONAL AND NONCONDITIONAL MUTANTS.

(Eng.) Bernstein, A. (Ontario Cancer Inst., 500 Sherbourne St., Toronto, Ontario M4X 1K9, Canada); MacCormick, R.; Martin, G. S. *Virology* 70(1): 206-209; 1976.

The "*x* segment" of the avian sarcoma viral genome is necessary for transformation of fibroblasts. Crosses between nonconditional transformation-defective mutants (lacking *x* segments; Rous sarcoma virus, subgroup b) and temperature-sensitive mutants (RSV, subgroup a) were made, to determine how many of the mutations resulting in temperature-sensitive transformation lie in the *x* segment. The results suggest that the RSV-b mutant results from a deletion close to the 3' end of the genome. Crosses between temperature-sensitive and transformation-defective mutants yielded transforming recombinants containing the entire genome. Tests of these recombinants by an infectious center technique suggest that the mutations in all of the temperature-sensitive viruses lie within the *x* segment.

3154 A CIRCULAR DNA-PROTEIN COMPLEX FROM ADENOVIRUSES AND ITS POSSIBLE ROLE IN DNA REPLICATION.

(Eng.) Robinson, A. J.; Bellett, A. J. D. In: *Tumor Viruses. Cold Spring Harbor Symposia on Quantitative Biology.* Cold Spring Harbor Lab. (Cold Spring Harbor, NY): vol. 39, pp. 523-531; 1975.

Experiments were performed, usually with chick embryo lethal orphan virus, to examine a model of adenovirus replication in which at some time the DNA is joined in a circle or concatemer. It was found that in the virus particle a tightly DNA-bound protein can join the end of the DNA. A general hypothesis for adenovirus DNA replication was proposed in which the replicating chromosomes are circular DNA-protein complexes that are converted to linear, genome-length molecules by pronase or sodium dodecyl sulfate (SDS) and alkali; synthesis is unidirectional; and the strands have different origins, one of which is the site of the SDS-pronase-induced break. The simplest model consistent with these proposals is a displacement loop mechanism. This hypothetical model explains how the 5' ends of the daughter strands are made, accounts for all the structures seen in SDS-pronase lysates and requires only one origin for each strand. (21 references)

3155 POLYAMINE METABOLISM IN NORMAL AND IN VIRUS-TRANSFORMED CHICK EMBRYO FIBROBLASTS.

(Eng.) Don, S. (Hebrew Univ.-Hadassah Medical Sch., Jerusalem, Israel); Bachrach, U. *Cancer Res* 35(12): 3618-3622; 1975.

The effect of transformation by Rous sarcoma virus on the ornithine decarboxylase levels of chick embryo fibroblasts was studied. Infection with Rous sarcoma virus resulted in increased ornithine decarboxylase activity (which preceded morphological alterations by at least 24 hr). Ornithine decarboxylase levels of normal controls, unlike those in Rous sarcoma virus-infected cells, declined with age; the enzyme activity of transformed cells was increased 20-fold 5 days postinfection. Infection with a temperature-sensitive mutant of Rous sarcoma virus, T5, stimulated ornithine decarboxylase activity of chick embryo fibroblasts at the permissive (37 C) but not at the nonpermissive (42 C) temperature. Ornithine decarboxylase activity of T5-infected cells (shifted from 42 C to 37 C); increased within 2 hr and reached the levels of wild-type, Schmidt-Ruppin-17A-transformed cells in 11 hr. When T5-transformed fibroblasts were shifted to the nonpermissive temperature, ornithine decarboxylase activity decreased fivefold within 4 hr and paralleled the levels of normal controls in 11 hr. Shifting of temperatures of incubation caused comparable alterations in cellular putrescine.

3156 CELL-SURFACE GLYCOPROTEINS IN NORMAL AND TRANSFORMED CELLS.

(Eng.) Warren, L. (Dept. Therapeutic Res., Univ. Pennsylvania Sch. Medicine, Philadelphia, PA 19174); Fuhrer, J. P.; Tuszynski, G. P.; Buck, C. A. *Biochem Soc Symp* 40: 147-157; 1974.

The proteins and glycoproteins extractable by various detergents from the water-insoluble cell protein fraction were investigated with the use of baby hamster kidney cells (BHK_{21}/C_{13}) as controls and their Rous virus-transformed counterpart (C_{13}/B_4). The cells were grown in the presence of ^{14}C-labeled amino acids and D-[^3H]glucosamine, harvested, homogenized, centrifuged, and pelleted. The pellet was extracted with lithium diiodosalicylate; then phenol was added and the extract was partitioned into an aqueous fraction and a phenol fraction. The two phases consisted of two separate populations of proteins and glycoproteins, as shown by chromatography on sodium dodecyl sulfate-hydroxyapatite. Preliminary work was done on the fractionation at the preparative level of the phenol phase of both groups of cells. A protein triplet in the 45,000-55,000 molecular wt range was observed in the middle of the gel. The middle band of the material from control cells was close to the upper band, whereas the three bands from the virus-transformed cells appeared to be equidistant. Milligram quantities of these bands have been obtained. When they were electrophoresed on sodium dodecyl sulfate-polyacrylamide gels, they appeared as single bands. Bands I and II from C_{13}/B_4 contained at least two proteins each, whereas the remainder appeared to be homogeneous. These studies suggest that there are differences (some possibly qualitative) in the hydrophobic membrane proteins of control and virus-transformed cells and that some proteins may be common to several membrane systems. (32 references)

3157 SYNTHESIS AND INTEGRATION OF VIRAL DNA IN CHICKEN CELLS AT DIFFERENT TIMES AFTER INFECTION WITH VARIOUS MULTIPLICITIES OF AVIAN ONCORNAVIRUS.

(Eng.) Khoury, A. T. (Rockefeller Univ., New York, NY 10021); Hanafusa, H. *J Virol* 18(2): 383-400; 1976.

In the attempt to find out if integration of the provirus resulting from RNA tumor virus infection is limited to specific sites in cell DNA, the variation in the number of virus-specific DNA copies produced and integrated in chicken embryo fibroblasts was determined at long and short intervals after high- and low-multiplicity Rous-associated virus 3 (RAV-2) infections and after several transfers. The number of copies of viral DNA in cells was determined by initial hybridization kinetics of single-stranded viral complementary DNA with a moderate excess of cell DNA. This hybridization accounted for the different sizes of cell DNA and complementary DNA in the hybridization mixture. Uninfected chicken embryo fibroblasts have approximately seven copies per haploid genome of DNA sequences homologous to part of the RAV-2 genome. Infection with RAV-2 added additional copies and different sequences of RAV-2-specific DNA. Between 13 and 20 hr after infection, there were 3-10 additional copies per haploid genome; this number remained constant despite increased multiplicity of infection. Between 20 and 40 hr postinfection, after some of the additional viral DNA was integrated, the cells accumulated up to 100 copies. Most of these copies were unintegrated. This number decreased with cell transfer, until cells were left with one to three copies of additional viral DNA sequences per haploid genome, of which most were integrated. The finding that viral infection caused the permanent addition of 1-3 copies of integrated viral DNA, despite the cells being confronted with up to 100 copies per haploid genome after infection, is consistent with a hypothesis that chicken cells contain a limited number of specific integration sites for the oncornavirus genome.

3158 ANOMALOUS VIRAL EXPRESSION IN RADIOGENIC LYMPHOMAS OF C57BL/KA MICE. (Eng.) Lieberman, M.; Kaplan, H. S.; Decleve, A. In: *Biology of Radiation Carcinogenesis*. Edited by J. M. Yuhas; R. W. Tennant; J. D. Regan. (New York: Raven Press): pp. 237-244; 1976.

An investigation of the time course of appearance and the location of radiation leukemia virus (RadLV) in the tissues of C57BL/Ka mice after a leukemogenic course of x-irradiation is presented. Although over 80% of C57BL/Ka thymocytes show the presence of viral antigens by 3 wk postinoculation, the thymocytes of x-irradiated mice remained essentially negative throughout lymphoma development. The replication characteristics of this virus in vitro were investigated. It was concluded that the radiation-induced lymphomas in C57BL/Ka mice differ strikingly from those induced by the passaged virus originally obtained from them; viral antigens are either absent or minimally detectable in the thymic lymphoma cells by the immunofluorescence procedure used. Irradiation of the mouse may lead to a derepression or activation of a latent viral genome, and afterward only a partial virus expression is needed to initiate transformation in susceptible cell types. An interesting feature, manifested after fibroblasts were infected in vitro with subcellular extracts from radiogenic lymphomas, was that the emergent virus can exhibit N-tropism, with single-hit kinetics on Fv-1n cells; passaged RadLV is distinctly B-tropic when adapted to growth in vitro. (9 references)

3159 EFFECT OF THE FV-1 LOCUS ON THE TITRATION OF MURINE LEUKEMIA VIRUSES. (Eng.) Jolicoeur, P. (Dept. Biology, Massachusetts Inst. Technology, Cambridge, MA 02139); Baltimore*, D. *J Virol* 16(6): 1593-1598; 1975.

The titration of N- and B-tropic murine leukemia viruses on sensitive and resistant cell lines was studied by direct XC plaque assay and infective center assay. The titration of a cloned B-tropic virus by infective center assay on BALB/3T3 ($Fv\text{-}1^{b/b}$) and NIH/3T3 ($Fv\text{-}1^{n/n}$) cells gave one-hit patterns (with 100-fold less infected NIH/3T3 cells than BALB/3T3 cells). Titration of B-tropic virus on DBA/2 cells ($Fv\text{-}1^{n/n}$) was also one-hit. The titration of a cloned N-tropic virus on NIH/3T3 and BALB/3T3 cells gave a one-hit curve; there were about 100-fold less infected BALB/3T3 cells than NIH/3T3 cells. Comparable results were obtained by titrating the cloned N-tropic virus on congenic SIM ($Fv\text{-}1^{n/n}$) and SIM.R ($Fv\text{-}1^{b/b}$) cells or the Gross N-tropic virus on BALB/3T3 cells. The multiple-hit phenomenon might not be an essential part of the Fv-1 gene restriction.

3160 THE MECHANISM OF REPLICATION OF ADENOVIRUS TYPE 5 DNA. (Eng.) Sussenbach, J. S.; Ellens, D. J.; van der Vliet, P. C.; Kuijk, M. G.; Steenbergh, P. H.; Vlak, J. M.; Rozijn, T. H.; Jansz, H. S. In: *Tumor Viruses. Cold Spring Harbor Symposia on Quantitative Biology*. Cold Spring Harbor Lab. (Cold Spring Harbor, NY): vol. 39, pp. 539-545; 1975.

The function of unbranched and branched intermediates in the replication of adenovirus type 5 DNA (Ad5 DNA), the localization of the origin of replication, and the mechanism of chain propagation were investigated. Analysis of replicating Ad5 DNA synthesized in KB cells within 30 min after the removal of 10^{-2} M hydroxyurea (HU) indicated that the first and second rounds of replication always start from the right-hand molecular end. The presence of branched intermediates with short single strands (down to 0.02 unit length) indicates that the origin of the displacement synthesis is positioned within 700 base pairs from the molecular right end. Viral DNA was annealed and the amount of single- and double-stranded DNA was determined by hydroxyapatite chromatography. Repeated analyses showed that the small pieces self-annealed for about 70%, suggesting that they originated from both complementary strands. The replication of both strands proceeds discontinuously. These pieces were not seen in the absence of HU, which means that the pieces joined very quickly under these conditions. The absence of circular structures from experimental preparations may be due to the isolation procedure or to the short lifetime of circular intermediates, or Ad5 DNA might have extraordinary molecular ends that permit replication of linear intermediates. The analysis of replicating Ad5 DNA suggests the presence, at the right end, of a unique series of nucleotides that can be recognized by an initiation factor. However, study of the molecular ends suggests a high degree of symmetry in the terminal nucleotide sequences of Ad5 DNA. (22 references)

3161 CELLULAR FACTORS THAT REGULATE RADIATION ACTIVATION AND RESTRICTION OF MOUSE LEUKEMIA VIRUSES. (Eng.) Tennant, R. W.; Otten, J. A.; Quarles, J. M.; Yang, W.-K.; Brown, A. In: *Biology of Radiation Carcinogenesis*. Edited by J. M. Yuhas; R. W. Tennant; J. D. Regan. (New York: Raven Press): pp. 227-236; 1976.

Cellular functions that are important in viral oncogenesis were investigated in AKR mouse embryo cell culture systems. Irradiation can activate the synthesis of infectious endogenous leukemia virus in cultured cells. Serum deprivation experiments suggested that cell division during irradiation is required for virus expression, or some event associated with cell division during exposure is required. A product from cells with the Fv-1 locus can transfer resistance to specific virus host-range types. The Fv-1 restriction probably functions primarily at some event in virus replication occurring within 6 hr after infection, and the Fv-1 locus specifies a unique mechanism of resistance to oncornaviruses that involves a gene product that functions intracellularly. (20 references)

3162 SYNTHESIS OF RAUSCHER MURINE LEUKEMIA VIRUS-SPECIFIC POLYPEPTIDES IN VITRO. (Eng.) Gielkens, A. L. (Dept. Biochemistry, Univ. Nijmegen, Geert Grooteplein Noord 21, Nijmegen, The Netherlands); Van Zaane, D.; Bloemers, H. P.; Bloemendal*, H. *Proc Natl Acad Sci USA* 73(2): 356-360; 1976.

The biosynthesis of virus-specific products was investigated. The biosynthesis of specific polypeptides directed by purified viral messenger RNA from JLS-V9 cells infected with Rauscher leukemia virus were studied in a rabbit reticulocyte lysate. The 35S viral mRNA gave rise to two major products of 65,000 and 72,000 molecular wt. The synthesis of specific polypeptides was also investigated in lysates derived from infected cells. The main products were polypeptides with molecular wt of 65,000, 76,000 and 82,000, and were preferentially made in association with membranes. The relative content of the virus-specific polypeptide of 65,000 molecular wt, synthesized in a cell-free system supplemented with purified polyribosomes, considerably higher for membrane-bound polyribosomes.

3163 THE POSSIBLE ROLE OF ONCORNAVIRUSES IN THE ETIOLOGY AND PATHOGENESIS OF MURINE LUPUS. (Eng.) Lerner, R. A. (Scripps Clinic and Res. Foundation, La Jolla, CA 92037); Dixon, F. J.; Croker, B. P.; Del Villano, B. C.; Jensen, F. C.; Kennel, S. J.; McConahey, P. J. *Adv Biosci* 12: 356-365; 1974.

The endogenous oncornavirus of New Zealand mice, the Scripps leukemia virus (SLV), was isolated from 25 different continuously growing lymphocyte cell lines, and its physical properties and biological potential were determined. Three months after infection with SLV, the sera of infected mice contained as much as 7.8 μg of rapidly turning over gs-1 protein/ml as determined by radioimmunoassay. The pathological effects of SLV were tested in newborn (NZB x BALB/c)F_1 and (BALB/c x NZB)F_1 mice. When these mice were injected with purified SLV, they developed leukemia-lymphoma, began to synthesize antinuclear antibodies, and often developed renal disease. Several features of the lymphocyte cell lines established from the New Zealand mice are of interest: (a) even though almost all of the cell lines were established from the spleen, all the cells from each established culture were of thymic origin (θ antigen was present on their surface), indicating that a selective mechanism is at work, since the starting material should contain about 50% B and 50% T cells; and (b) in every cell line established, a marker chromosome was present, and often a deletion of the distal portion of one of the X chromosomes was also seen. It is suggested that the presence of these marker chromosomes could also be due to selection of certain cells from mosaic mice or could be a nonrandom genetic event that occurred in tissue culture. (6 references)

3164 DELETION MAPPING OF MOLONEY TYPE C VIRUS: POLYPEPTIDE AND NUCLEIC ACID EXPRESSION IN DIFFERENT TRANSFORMING VIRUS ISOLATES. (Eng.) Parks, W. P. (Lab. of Tumor Virus Genetics, Natl. Cancer Inst., Bethesda, MD 20014); Howk, R. S,; Anisowicz, A.; Scolnick, E. N. *J Virol* 18(2): 491-503; 1976.

Because each of the genomes responsible for fibroblast transformation (FT) appears to be a recombinant with part of the genome of Moloney murine leukemia virus (Mo-MuLV), the Mo-MuLV nucleic acid sequences remaining in each of the FT+ isolates were correlated with the Mo-MuLV structural polypeptides synthesized in transformed heterologous nonproducer cells. To measure the Mo-MuLV nucleic acid sequences, viral complementary DNA (cDNA) was fractionated by hybridization to RNA from different nonproducer cells. Because of the differing patterns of polypeptide expression, the transformed nonproducer cells were analyzed for RNA homologous to Mo-MuLV; in correlation with the polypeptide differences, different portions of the Mo-MuLV genome were expressed in these cells. Hybridization showed that a portion of the murine type C genome was present in all the replication-defective murine FT+ viruses. These results suggest that the recombination event leading to FT+ virus formation occurs at a common region of the murine type C viral genome. A correlation was also found between the final extents of Mo-MuLV RNA expressed and the number of structural polypeptides produced; this suggests that the cDNA probes prepared by cycling against different nonproducer RNAs represent at least part of the sequences coding for p30 and/or the low-molecular-wt proteins. That the patterns of RNA or protein expression never changed in the transformed nonproducer cells indicates that these FT+ viruses are deletion mutants and that the RNA and protein expression patterns noted are due to differing deletion of the Mo-MuLV genome and not to transcriptional or translational blocks. Deletion mapping of Mo-MuLV is possible and, by correlating the proteins synthesized with the RNA expressed, one can prepare cDNA probes representing different portions of the Mo-MuLV genome.

3165 FRACTIONATION OF DNA NUCLEOTIDE TRANSCRIPTS FROM MOLONEY SARCOMA VIRUS AND ISOLATION OF SARCOMA VIRUS-SPECIFIC COMPLEMENTARY DNA. (Eng.)

Frankel, A. E. (Lab. Viral Carcinogenesis, Natl. Cancer Inst., Bethesda, MD 20014); Neubauer, R. L.; Fischinger, P. J. *J Virol* 18(2): 481-490; 1976.

Radioactive DNA complementary to nucleotide sequences in Moloney murine sarcoma virus (MSV) and Moloney leukemia virus (M-MuLV) complex was made by the endogenous reverse transcriptase reaction. According to biological assay, these virus stocks contained a threefold excess of MSV over M-MuLV. The complementary DNA (cDNA) was an accurate copy of the viral RNA because 86% of 35S viral RNA hybridized with complementary (cDNA) DNA at a 1.5 to 1 cDNA-RNA molar ratio (hybridization was used to verify the presence of the sarcoma-specific sequences). The cDNA, of a 4-6S size, was fractionated by sequential absorptions with MuLV and the feline leukemia virus pseudotype of MSV, [MSV(FeLV)] RNA. In this manner three sets of nucleotide sequences, which represent different portions of the MSV viral complex, were obtained: a sarcoma virus-specific fraction (cDNAsarc) with sequences that had no homology to M-MuLV RNA but which hybridized to MSV(FeLV) RNA; a sarcoma-leukemia fraction (cDNAcommon) with sequences common to MSV as well as M-MuLV viral RNA; and a cDNAleuk representing those nucleotide sequences found only in M-MuLV. Hybridization of MSV-MuLV viral 35S RNA with a threefold molar excess of cDNA's revealed that approximately 20% was hybridized with cDNAsarc, whereas approximately 75% was hybridized with cDNAcommon. M-MuLV 35S RNA alone did not hybridize with cDNAsarc but did hybridize 40% and 50% with cDNAleuk and cDNAcommon, respectively. The cDNAsarc represented about 25% of the total MSV sequences, whereas the cDNAcommon represented the remainder of the MSV virus genome. Some cDNAcommon sequences were shared by two other sarcoma viruses and several distinctly different isolates of MuLV. In contrast, the MSV ''sarc'' sequences had little or no homology with two other murine sarcoma virus isolates.

3166 RNA METABOLISM OF MURINE LEUKEMIA VIRUS III. IDENTIFICATION AND QUANTITATION OF ENDOGENOUS VIRUS-SPECIFIC mRNA IN THE UNINFECTED BALB/C CELL LINE JLS-V9. (Eng.)

Fan, H. (Tumor Virology Lab., Salk Inst., San Diego, CA 92112); Mueller-Lantzsch, N. *J Virol* 18(2): 401-410; 1976.

In the analysis of JLS-V9 cell (an uninfected BALB/c-derived cell line) cytoplasmic subfractions, the amount of virus-specific RNA in different JLS-V9 cell fractions was quantitated in comparison to cell fractions obtained from M-MuLV clone no. 1 cells (a line of NIH 3T3 cells producing Moloney murine leukemia virus). Quantitation was performed by annealing extracted RNA with ^{3}H-labeled virus-specific DNA. Approximately 40% of the total virus-specific mRNA was recovered in the purified polyribosomes in M-MuLV no. 1 cells. The amount of virus-specific RNA on polyribosomes appeared to be similar for JLS-V9 cells and M-MuLV clone no. 1 cells (10% and 5.6%, respectively). In contrast, the level of virus-specific protein in JLS-V9 cells (monitored by radioimmunoassay of the internal structural protein p30) was less than 2% the level in the M-MuLV clone no. 1 cells. The finding that JLS-V9 cells contained relatively high levels of virus-specific mRNA but very little virus-specific protein may be explained by one of three possible ways: (a) virus-specific proteins may be synthesized in the cells at relatively normal rates, but, because they are unstable, they are quickly regraded; (b) precursor proteins for the virus-specific proteins exist in the cells but are not detectable by normal enzymatic and immunologic techniques; (c) ribosomes are present on virus-specific mRNA but translation from them is very inefficient.

3167 A LABORATORY MODEL FOR THE STUDY OF THE IMMUNOBIOLOGY OF OSTEOSARCOMA. (Eng.)

Friedlaender, G. E. (Yale Univ. Sch. Medicine, 333 Cedar St., New Haven, CT 06510); Mitchell, M. S. *Cancer* 36(5): 1631-1639; 1975.

The inoculation of Moloney sarcoma virus into the marrow cavity of the tibia of newborn Wistar-Lewis rats resulted in the appearance of an initially localized osteosarcoma in 97.7% of the animals. At least 77.9% of the rats developed lung metastases and died (usually within 6 wk of inoculation). The remaining 22.1% showed regression of disease after the initial growth of the tumor. Tumor cells were maintained in tissue culture and used as target cells for a visual and isotopic (^{3}H-thymidine or ^{125}IUdR) microcytotoxicity assay. Cell-mediated immunity could be measured by these methods throughout the course of the illness in animals with progressive disease as well as in those whose tumors eventually regressed. The Moloney-sarcoma-virus-induced rat osteosarcoma and human osteosarcoma appeared to have several basic pathologic and immunologic similarities. The model could be useful for studying the effects of a variety of treatment protocols upon the clinical course and immune response to osteosarcoma.

3168 CHARACTERIZATION OF DEFECTIVE SIMIAN VIRUS 40 DNA: COMPARISON BETWEEN LARGE-PLAQUE AND SMALL-PLAQUE TYPES. (Eng.)

Yoshiike, K.; Furuno, A.; Watanabe, S.; Uchida, S.; Matsubara, K.; Takagi, Y. In: *Tumor Viruses. Cold Spring Harbor Symposia on Quantitative Biology.* Cold Spring Harbor Lab. (Cold Spring Harbor, NY): Vol 39, pp. 85-93; 1975.

The changes resulting from repeated propagation of viruses at high multiplicities of infection were studied, by electron microscopy, in the defective DNA molecules of large and small-plaque SV40 produced and accumulated during serial undiluted passages. Defective SV40 DNA contained deletions, insertions, and substitutions. The majority of the large-plaque defective molecules had a simple deletion. The sizes of the deletions were heterogeneous, and the sites of deletion appeared to be mostly overlapping. The majority of the small-plaque defective molecules had a deletion, and more than half of the deletion molecules also had an insertion at a separate site. The small-plaque SV40 genome appeared to have more sites for deletion than the large-plaque SV40 genome, and the deletions occurred at more than two sites. The change of a local nucleotide sequence resulting from mutation may be related to the difference between the two plaque types.

3169 SPECIFIC REITERATION OF VIRAL DNA SEQUENCES IN MAMMALIAN CELLS. (Eng.) Martin, M. A.; Khoury, G.; Fareed, G. C. In: *Tumor Viruses. Cold Spring Harbor Symposia on Quantitative Biology.* Cold Spring Harbor Lab. (Cold Spring Harbor, NY): vol. 39, pp. 129-136; 1975.

A unique variant of simian virus 40 (SV40) DNA that consists of a triplication of a specific one-third of the virus genome was characterized. Host cell DNA could not be found in such reiterated molecules. An SV40-like virus, originally isolated from the brain of a patient with progressive multifocal leukoencephalopathy, was found to contain DNA that was virtually identical to SV40 DNA. After several passages, a supersensitive DNA species was produced by *Escherichia coli* restriction endonuclease (R.*Eco*RI) cleavage which sedimented at 10.4S. These 10.4S molecules contain three R.*Eco*RI cleavage sites and represent a tandem triplication of a specific one-third of the parental virus. Of 300 molecules examined electron microscopically, only one had two replication loops and none had three. The emergence of triplicated viral genomes may occur by a recombination event similar to the one that takes place during the formation of adeno-SV40 hybrid virions. (20 references)

3170 INTEGRATION OF ADENOVIRUS DNA INTO THE CELLULAR GENOME. (Eng.) Doerfler, W.; Burger, H.; Ortin, J.; Fanning, E.; Brown, D. T.; Westphal, M.; Winterhoff, U.; Weiser, B.; Schick, J. In: *Tumor Viruses. Cold Spring Harbor Symposia on Quantitative Biology.* Cold Spring Harbor Lab. (Cold Spring Harbor, NY): vol. 39, pp. 505-521; 1975.

The results from two different experimental systems concerning the integration of adenovirus (Ad) DNA into the cellular genome are presented. KB cells productively infected with Ad2 contain a fast-sedimenting form of viral DNA (50S-90S) that is stable in alkali; this represents viral DNA integrated into the host DNA. Reassociation experiments suggest that about 2,000-5,000 viral DNA copies per cell exist in the fast-sedimenting form. The 50S-90S DNA does not reanneal appreciably with itself. After the purified viral DNA was extracted from purified Ad2 virions, circular forms of viral DNA were seen by electron microscopy; these circles represent the total genome or one-quarter of the viral genome. These circles may serve as intermediates in the integration process. The transcription kinetics of Ad12 DNA in abortively infected BHK21 cells was determined. RNA molecules are found that sediment at 25S and at 35S in sucrose density gradients; viral RNA of high molecular wt may be transcribed from integrated viral sequences. There are palindromic sequences in the Ad2 genome; these palindromes may serve as viral counterparts of repetitive or palindromic sequences in mammalian DNA that may serve as the recognition system required for integration. (48 references)

3171 FORMATION OF REITERATED SIMIAN VIRUS 40 DNA. (Eng.) Davoli, D.; Fareed, G. C. In: *Tumor Viruses. Cold Spring Harbor Symposia on Quantitative Biology.* Cold Spring Harbor Lab. (Cold Spring Harbor, NY): vol. 39, pp. 137-146; 1975.

The amplification of reiteration of specific viral DNA sequences was studied. The one-third DNA segments (10.4S), after *Escherichia coli* endonuclease I cleavage of the triplication mutant DNA, were isolated, and the fate of these closed circular molecules (minicircles) was examined. When the minicircular DNA was used to infect monkey cells, its replication was not detected. After the cells were infected with the minicircles and simian virus 40 (SV40) DNA, tritiated thymidine was incorporated into both viral DNA species. Furthermore, 26% of the tritiated superhelical DNA sedimented more rapidly than SV40 DNA I (21S) in neutral sucrose gradients (22S-32S). A similar amount of this rapidly sedimenting DNA was also found when intact DAR (a human variant of SV40) DNA containing the triplication mutant was tested. Endonuclease cleavage of this DNA produced 10.4S segments. Cleavage of the 21S DNA also produced 10.4S DNA. These results indicate that the minicircle molecules are amplified in vivo, yielding the original triplication mutant and a heterogeneous oligomers population in which the 10.4S segment is reiterated as many as 6 to 9 times. These minicircular segments lack essentially all of the early gene region of SV40. (24 references)

3172 ISOLATION, PROPAGATION AND CHARACTERIZATION OF REPLICATION REQUIREMENTS OF REITERATION MUTANTS OF SIMIAN VIRUS 40. (Eng.) Ganem, D. (Harvard Medical Sch., Boston, MA 02115); Nussbaum, A. L.; Davoli, D.; Fareed, G. C. *J Mol Biol* 101(1): 57-83; 1976.

Five reiteration mutants, in which a large segment of the simian virus 40 (SV40) genome has been deleted and the remaining viral DNA segment (monomer) is tandemly reiterated, have been identified from serially propagated, defective stocks of SV40 and DAR virus (a human SV40-like virus). The monomer DNA segments from the mutant genomes were separated from the wild-type SV40 DNA after cleavage by the *Escherichia coli* restriction endonuclease RI or by the *Hemophilus influenzae* d restriction endonuclease, which produce short cohesive termini at their cleavage sites. These monomer segments, which are 1/3, 1/4, or 1/5 the size of wild-type SV40 DNA, were then circularized in vitro with use of bacteriophage T4 polynucleotide ligase and used to infect African green monkey kidney cells in the presence of wild-type or temperature-sensitive (ts) mutant DNAs as helpers. Early ts mutant genomes were unable to serve as helpers in the replication and amplification of these minicircular DNAs at the nonpermissive temperature. These DNAs were amplified in vivo through an arithmetic series of oligomers. Encapsidation of reiterated molecules between 70%-100% the size of wild-type SV40 DNA was observed, although reiterated viral DNA molecules much larger than unit size were found in vivo. The requirement of all five mutants for helper genomes for replication suggests that the initiation process of DNA replication in these mutants proceeds by the same mechanism as in wild-type SV40. This, in turn, suggests that the monomer segment retains the SV40 initiation site for DNA replication; the reiterant, therefore, must contain multiple copies of this site. It seems likely that this reiteration of the replication origin is the structural feature that underlies the tremendous growth advantage of the reiterant over the helper DNA. Since these reiteration mutants contain monomer segments capable of replication and amplification in vivo in the presence of helper genomes, they may be particularly valuable as vehicles for propagating foreign DNA in mammalian cells. (53 references)

3173 NUCLEOTIDE SEQUENCE ABOUT THE 3' TERMINUS OF SV40 DNA TRANSCRIPTS AND THE REGION WHERE DNA SYNTHESIS IS INITIATED. (Eng.) Dhar, R.; Subramanian, K.; Zain, B. S.; Pan, J.; Weissman, S. M. In: *Tumor Viruses. Cold Spring Harbor Symposia on Quantitative Biology*. Cold Spring Harbor Lab. (Cold Spring Harbor, NY); vol. 39, pp. 153-160; 1975.

Information concerning the sequences between the 3' ends of early and late messenger RNA, the sequences at one of the sites at which simian virus 40 (SV40) DNA interacts with *Escherichia coli* RNA polymerase, and the sequences at the site of initiation and termination of DNA synthesis is reviewed. The early region of SV40 DNA may be slightly longer than previously supposed; it is more than 40% of the total molecule (well over 2,000 base pairs). It was suggested that at the 3' end at least 50 nucleotides lie beyond the last terminator codon; this may be a common eukaryotic feature. Either termination of transcription or cleavage of precursor RNA occurs very near to or at the identical site recognized by *E. coli* RNA polymerase and *Hemophilus influenzae* restriction endonuclease. Analysis of the region about the origin of DNA replication indicates several potential sites for recognition and interaction with specific proteins; there may be a partial resemblance between certain sites at the origin and termination of DNA replication in wild-type SV40. (34 references)

3174 GENETICS OF CELL TRANSFORMATION BY SV40. (Eng.) Croce, C. M. In: *Biology of Radiation Carcinogenesis*. Edited by J. M. Yuhas; R. W. Tennant; J. D. Regan. (New York: Raven Press): pp. 207-215; 1976.

Hybridization studies of normal mouse peritoneal macrophages with simian virus 40 (SV40)-transformed human cells indicated that the hybrids were SV40 T antigen-positive in 168/168 cases, indicating that the human chromosome carrying the SV40 genome was retained by all the hybrids. Karyological analysis in 67 hybrids confirmed the results. None of the hybrid cell clones examined displayed the density-dependent inhibition of cell division of normal cells. Hybrids had a saturation density five times higher than that of normal diploid human cells and four times higher than that of mouse fibroblasts. Hybrids gave rise to colonies in soft agar, a characteristic of transformed cells; thus, the dominance of the transformed phenotype is confirmed in the hybrids. It is inferred that the human chromosome 7 carrying the SV40 genome contains gene(s) coding for transforming gene products and that chromosome 7 is responsible for the growth of the hybrid cells, since no segregant cells were found. These hybrids were tumorigenic when injected into nude mice, and karyological analysis of resulting tumor cells indicated that they carried the full complement of mouse chromosomes and only a very few human chromosomes; the human chromosome 7, however, was included. Hybridization of nondividing mouse cells with human cancer cells could result in the retention of a specific human chromosome responsible for the expression of the cancer phenotype, thus allowing identification of the human chromosome(s) responsible for the phenotype. (18 references)

3175 ELECTRON MICROSCOPIC MAPPING OF THE ATTACHMENT SITES ON SV40 DNA DURING LYTIC INFECTION. (Eng.) Chow, L. T.; Boyer, H. W.; Tischer, E. G.; Goodman, H. M. In: *Tumor Viruses. Cold Spring Harbor Symposia on Quantitative Biology*. Cold Spring Harbor Lab. (Cold Spring Harbor, NY): vol. 39, pp. 109-117; 1975.

More than 20 different preparations of simian virus 40 (SV40) linear DNA molecules of unique sequence generated by bacterial restriction endonucleases were studied by electron microscopy. Membrane filter hybridization techniques were also used. It was shown that there are one major and two minor sites on SV40 where substitutions occur. A preparation of SV40 DNA with a high frequency of substitutions contains reiterated host cell DNA, whereas a preparation with almost no substitutions does not. The results strongly support the hypothesis that SV40 DNA integrates semispecifically into host cell DNA during lytic infection. (30 references)

3176 NUCLEAR AND CYTOPLASMIC ADENOVIRUS RNA. DIFFERENCES BETWEEN 5'-TERMINI OF MESSENGER AND NON-MESSENGER TRANSCRIPTS. (Eng.) McGuire, P. M. (Yale Univ. Sch. Medicine, 310 Cedar St., New Haven, CT); Piatak, M.; Hodge, L. D. *J Mol Biol* 101(3): 379-396; 1976.

The 5'-terminus of adenovirus-specific RNA in nuclear hnRNA (rapidly sedimenting), polyribosome-associated mRNA (slowing sedimenting cytoplasmic RNA), and in nuclear and cytoplasmic viral-associated RNA (VA-RNA) was examined late in the infection of HeLa cells. Two types of 5'-termini were isolated. A purine tetraphosphate (pppGp) was observed in both nuclear and cytoplasmic VA-RNA as well as in apparent viral nuclear-precursor RNA, which also contained an inverted dinucleotide. Viral transcripts containing poly(A) segments, whether from the nuclear hnRNA or from polyribosome-associated RNA, had an inverted dinucleotide(s) only at the 5'-terminus. These results bear on aspects of the posttranscriptional metabolism of viral nuclear RNA, especially if the nuclear RNA sedimenting more rapidly than 32S hnRNA is considered to contain precursor mRNA sequences. The structures isolated from this nuclear hnRNA suggest a possible sequence of events in processing. This RNA contained both types of 5'-termini, but when further selected for transcripts with poly(A) segments only, an inverted dinucleotide was present. Modification of 5'-ends in at least some precursor molecules, therefore, may occur before complete cleavage to polyribosome-size species and before addition of poly(A) sequences to 3'-ends. It is suggested that only virus-specific transcripts destined to function as cytoplasmic messenger RNA undergo modification at their 5'-termini before exit from the nucleus. The exact sequence of the 5'-ends in both nuclear hnRNA and polyribosome-associated RNA, as well as the number of mRNA transcripts with blocked 5'-ends, remains to be clarified. (49 references)

3177 RNA SYNTHESIS AND PROCESSING IN ADENOVIRUS-INFECTED CELLS. (Eng.) Philipson, L.; Pettersson, U.; Lindberg, U.; Tibbetts, C.; Vennstrom, B.; Persson, T. In: *Tumor Viruses. Cold Spring Harbor Symposia on Quantitative Biology.* Cold Spring Harbor Lab. (Cold Spring Harbor, NY): vol. 39, pp. 447-456; 1975.

RNA synthesis and processing in the adenovirus system were investigated. Adenovirus type 2 (Ad2) was propagated in suspension cultures of HeLa-53 or KB cells, RNA was extracted, RNA-DNA hybrids were formed, and filter hybridization of viral RNA onto viral DNA was performed. In the nucleus, large heterogeneous viral RNA molecules were detected, some of which correspond to about half the size of one adenovirus DNA strand. It appears likely that polyadenylation is necessary for proper transport of viral RNA to the cytoplasm. Viral sequences may be synthesized preferentially late after infection, leading to an accumulation of viral mRNA in the cytoplasm. Late transcription does not appear to be symmetric. (42 references)

3178 CHARACTERIZATION OF THE DNA FROM INCOMPLETE PARTICLES OF ADENOVIRUS TYPE 2. (Eng.) Tjia, S. (Institut fuer Genetik der Universitaet zu Koeln, Weyertal 121, D-5000 Koeln 41, W. Germany); Fanning, E.; Schick, J.; Doerfler, W. *Hoppe Seylers Z Physiol Chem* 357(3): 339-340; 1976.

DNA from incomplete adenovirus type 2 particles from productively infected KB cells was characterized by DNA-DNA filter hybridization to cell and viral DNA. The DNA sedimenting between 5S and 25S was used. In addition, the segments of viral DNA from the incomplete particles were determined by reassociation kinetic experiments using unlabeled DNA from the particles and each of the six ^{3}H-labeled fragments of viral DNA cleaved by *Eco*RI restriction endonuclease. In other experiments, labeled DNA from the particles was reassociated with an excess of cellular DNA. The results indicated that the incomplete particles contained both viral and cellular DNA. All fractions of the viral genome were represented in about equal amounts.

3179 CELL TRANSFORMATION: A STUDY OF REGULATION WITH TYPES 5 AND 12 ADENOVIRUS TEMPERATURE-SENSITIVE MUTANTS. (Eng.) Ginsberg, H. S.; Ensinger, M. J.; Kauffman, R. S.; Mayer, A. J.; Lundholm, U. In: *Tumor Viruses. Cold Spring Harbor Symposia on Quantitative Biology.* Cold Spring Harbor Lab. (Cold Spring Harbor, NY): vol. 39, pp. 419-426; 1975.

The types 5 and 12 temperature sensitive (ts) mutants of adenovirus (Ad) that cannot replicate their DNA under nonpermissive conditions (39.5 C) are described. In addition, one that can make the major viral gene products but under nonpermissive conditions cannot transport its hexon protein into the nucleus for virion assembly is characterized. The following insights were achieved: (a) viral DNA replication requires at least two specific viral proteins; (b) hexon protein transport requires a nonhexon viral protein; (c) virions assembly cannot ensue if one of the viral gene products is defective; and (d) transformation is the consequence of abortive infections with many adenoviruses, but its incidence is increased after infection with one class of adenovirus ts mutants. Two models are presented to explain the last observation. In one model, the DNA-binding protein normally maintains the viral DNA in a specific conformation or attached to some cellular structure so that viral genome integration is prohibited and viral DNA replication is initiated and continues. In the absence of this protein integration is more commonly permitted. In the second model, viral genome integration into a host chromosome is a normal event in the initial stages of infection, even during a productive infection. The latter hypothesis suggests that in a normal productive infection the entire viral genome is excised and replicated: defective excision occurs in abortively infected cells, leaving genome fragments to initiate and maintain transformation. (40 references)

3180 TRANSCRIPTION OF 70S RNA BY DNA POLYMERASES FROM MAMMALIAN RNA VIRUSES. (Eng.) Abrell, J. W. (Natl. Cancer Inst., Bethesda, MD 20014); Reitz, M. S.; Gallo*, R. C. *J Virol* 16(6): 1566-1574; 1975.

The DNA polymerases purified by the same procedure from four mammalian RNA viruses (simian sarcoma virus type 1, gibbon ape lymphoma virus, Mason-Pfizer monkey virus, and Rauscher murine leukemia virus) were capable of transcribing heteropolymeric regions of viral 70S RNA without any other primer. In this reconstituted system, the enzymes from simian sarcoma virus type 1, Mason-Pfizer monkey virus, and Rauscher murine leukemia virus transcribed viral 70S RNA almost as efficiently as the DNA polymerase from the avian myeloblastosis virus; however, gibbon ape lymphoma virus DNA polymerase was approximately 3- to 5-fold less efficient. Although there was a substantial difference among the sizes of these DNA polymerases (160,000 daltons for the avian myeloblastosis virus enzyme, 110,000 daltons for the Mason-Pfizer monkey virus enzyme, and 70,000 daltons for the mammalian type C viral polymerases), the ability to transcribe viral 70S RNA was a common characteristic.

3181 HERPES SIMPLEX VIRUS-SPECIFIC ANTIGENS IN EXFOLIATED CERVICAL CELLS FROM WOMEN WITH AND WITHOUT CERVICAL ANAPLASIA.

(Eng.) Pacsa, A. S. (County Hosp., Pecs, Hungary 7643); Kummerlander, L.; Pejtsik, B.; Krommer, K.; Pali, K. *Cancer Res* 36(7): 2130-2132; 1976.

The presence of herpes simplex virus (HSV)-specific antigens was investigated by indirect immunofluorescence in the cervical cells of 530 women with normal cervical epithelium, 175 with bland disorders (including ectopium, iodine-negative areas, erosion, inflammations, and histological diagnoses such as keratosis, hyperplasia, metaplasia, and basal activity), 52 with dysplasias, and 38 with invasive cervical carcinomas of the uterine cervix. Antigens were present in 9% of samples from women with normal cervical epithelium; they were present in 41% of the samples from women with bland disorders, 61% of those from dysplasia patients, and 94% of those from invasive carcinoma patients. The testing of 3 consecutive imprints of 68 antigen-positive and 232 antigen-negative women at 6-mo intervals revealed that, in cervical cells, HSV-specific antigens persisted throughout the 1-yr followup period. The results presented support the possibility that cervical dysplasias or tumors originate from antigen-carrying cells. (15 references)

3182 THE RELATION OF THE MITOGENIC ACTION OF POLYOMA AND RELATED VIRUSES TO TUMOR FORMATION.

(Eng.) Weil, R. (Dept. Molecular Biology, Univ. Geneva, 1211 Geneva 4, Switzerland); May, E.; May, P. *Adv Biosci* 12: 579-596; 1974.

Polyoma (Py) and related viruses may act as endogenous mitogens. The molecular mechanism of their mitogenic action and its relation to virus-induced cell transformation and tumor formation in vivo are discussed. The studies were performed in confluent primary mouse kidney (MK) cell cultures consisting mainly of epitheloid cells in phase G_0. The sequence of events taking place during abortive infection with SV40 indicates that early SV40-specific messenger RNA contains information necessary for the production of SV40-specific T antigen and that this antigen (or an unknown early viral product the synthesis of which may parallel the appearance of T antigen) activates a cellular regulatory element that controls chromosome replication and mitosis. Under standard conditions of infection, most cells in primary MK cultures undergo only one cycle of SV40-induced mitosis. On a small fraction of the abortively infected MK cells, however, the infection confers a continuous mitotic stimulus and, apparently related to it, the ability to resume the synthesis of SV40-specific early RNA and of T antigen after · mitosis. SV40-induced mitotic stimulus and cytogenic variation lead within a few weeks to the conversion of the primary MK cultures into continuous cell lines. These early transformed cells are not malignant; SV40-induced malignant transformation of mouse tissue culture cells appears to be a relatively slow, secondary process involving cytogenetic variation and selection. Light and electron microscopy studies show that infection with Py in newborn hamsters and rabbits induces a widespread, intense mitotic stimulation, and that many Py-induced tumors may be noninvasive and nonmetastasizing hyperplasias. All tumor cells contained intranuclear Py-specific T antigen. The mitogenic effect of Py observed in tissue culture cells may also be operational in vivo, leading in newborn hamsters and rats to the development of hyperplasias. The development of cancer appears to be the result of secondary events. The viral genome is apparently not required to maintain the malignant phenotype once it has been established. Although viruses may play a role in the development of at least certain types of human cancer, it cannot be assumed that all malignancies are simply the phenotypic expression of integrated viral cancer genes or of activated oncogenes. (60 references)

3183 ANTIBODIES TO EPSTEIN-BARR VIRUS IN PATIENTS WITH HODGKIN'S DISEASE AND LEUKEMIA.

(Eng.) Gotlieb-Stematsky, T. (Central Virus Lab., Ministry Health, Tel-Aviv-Jaffa, Israel); Vonsover, A.; Ramot, B.; Zaizov, R.; Nordan, U.; Aghai, E.; Kendo, C.; Modan, M. *Cancer* 36(5): 1640-1645; 1975.

The sera from 67 Hodgkin's disease patients, 71 leukemia patients, and 186 healthy subjects were tested for antibodies to Epstein-Barr (EB) viral antigens by immunofluorescence methods. In both disease categories, in particular Hodgkin's disease patients, levels of antibodies to the viral capsid antigen (EBV-VCA) were higher than in the healthy controls. Significantly higher titers were found in Jewish patients of Asian-African origin (as compared to Jews of European origin, with Arab patients as intermediates). The effect of ethnic origin was independent of age and histopathologic type. Sex had no effect on titer. Inconsistent differences in titer were found between age groups in the various ethnic-histopathologic type groups. Some of the leukemia patients had no detectable antibodies to EBV, while all Hodgkin's disease patients showed previous contact with EB virus. Antibodies to the early antigen (EBV-EA) were found in 27% of Hodgkin's and 37% of leukemia patients.

3184 EFFECT OF SERUM CONCENTRATION ON ADENYLATE CYCLASE ACTIVITIES OF UNTRANSFORMED BHK CELLS AND POLYOMA VIRUS-TRANSFORMED DERIVATIVES.

(Eng.) Matsumoto, T. (Sapporo Medical Coll., Minami-1-jo, Nishi-17-chome, Chuo-ku, Sapporo 060 Japan); Uchida, T. *Gann* 66(4): 393-397; 1975.

The adenylate cyclase activities of untransformed hamster BHK 21/13 cells and their polyoma virus-transformed derivatives were compared. The untransformed cells cultured in a medium containing 10% serum had a slightly higher activity than the transformed cells. When the cells were cultured in a medium containing 0.5% serum that stopped proliferation of untransformed cells, greater significant difference was observed in the adenylate cyclase activity; the untransformed cells had a 2-fold higher activity than the transformed cells. The full activity measured in the presence of fluoride was 2-fold higher in the untransformed than in the transformed cells. The low serum concentration increased the enzyme activity of both untransformed and transformed cells; however, the enzyme activity of transformed cells in the presence of 0.5% serum did not exceed the activity of untransformed cells in 10% serum. Intracellular cyclic AMP levels were in parallel with the activity of adenylate cyclase.

3185 ANALYSIS OF PLATELETS FROM PATIENTS WITH THROMBOCYTHEMIA FOR REVERSE TRANSCRIPTASE AND VIRUS-LIKE PARTICLES. (Eng.) Brodsky, I. (Hahnemann Medical Coll. and Hosp., Philadelphia, PA 19102); Fuscaldo, A. A.; Erlick, B. J.; Kingsbury, E. W.; Schultz, G. M.; Fuscaldo, K. E. *J Natl Cancer Inst* 55(5): 1069-1074; 1975.

The preliminary analysis of an RNA-directed DNA polymerase was made, and a C-type virus-like particle was identified in platelets from two patients with thrombocythemia (primary, essential, hemorrhagic, or idiopathic thrombocythemia). Platelet homogenates were centrifuged through a sucrose equilibrium density gradient. Both endogenous and exogenous DNA polymerase activity was found at a density of 1.19 g/ml. In control gradients, no activity was seen at comparable densities. Electron micrographs of thin sections of these platelets revealed a particle with the morphologic characteristics of a C-type virus. The diameter of this particle was about 80 nm (slightly lower than that commonly found for C-type particles). Critical-point dried specimens, from the fractions of the sucrose gradient at which DNA polymerase activity was found, contained particles of the same morphology and size as those in the thin sections.

3186 CATALYTIC SYNTHESIS OF cDNAs BY RNA-DIRECTED DNA POLYMERASE (Proceedings Abstract). (Eng.) Krueger, L. J. (Molecular Hematology Branch, NHLI, NIH, Bethesda, MD 20014); Weiss, G. B.; Merrick, W. C.; Anderson, W. F. *Fed Proc* 35(7): 1592; 1976.

3187 35S A AND B RNA SUBUNITS OF AVIAN RNA TUMOR VIRUS STRAINS CLONED AND PASSAGED IN CHICK AND DUCK CELLS. (Eng.) Stone, M. P.; Smith, R. E.; Joklik, W. K. In: *Tumor Viruses. Cold Spring Harbor Symposia on Quantitative Biology*. Cold Spring Harbor Lab. (Cold Spring Harbor, NY): Vol 39, pp. 859-868; 1975.

3188 ISOLATION OF VIRUS-INDUCED ANTIGENS FROM CELLS INFECTED WITH MAREK'S DISEASE AND TURKEY HERPESVIRUSES. (Eng.) Kaaden, O. R.; Dietzschold, B. In: *Oncogenesis and Herpesviruses II. Part 2: Epidemiology, Host Response and Control. Proceedings of a Symposium held in Nuremberg, Federal Republic of Germany, 14-16 October 1974*. International Agency for Res. on Cancer. (Lyon, France): pp. 337-344; 1975.

3189 A NEW REVERSE TRANSCRIPTASE IN THE CHICKEN SYSTEM NOT RELATED TO VIRUS (Abstract). (Eng.) Bauer, G. (Max-Planck-Institut fuer Biochemie, Abt. Virusforschung, D-8033 Martinsried, W. Germany); Jilek, G.; Hofschneider, P. H. *Hoppe Seylers Z Physiol Chem* 357(3): 307-308; 1976.

3190 EXPRESSION OF TUMOR SPECIFIC AND EMBRYONIC ANTIGENS ON AVIAN ONCORNAVIRUS TRANSFORMED CHICKEN AND MOUSE CELLS. (Eng.) Kurth, R. (Robert Koch-Institut, Abteilung fuer Virologie, 1 Berlin 65, W. Germany); Bauer, H. *Adv Biosci* 12: 479-494; 1974.

3191 tRNAs ASSOCIATED WITH THE 70S RNA OF AVIAN MYELOBLASTOSIS VIRUS. (Eng.) Waters, L. C. (Biology Div., Oak Ridge Natl. Lab., Oak Ridge, TN 37830); Mullin, B. C.; Bailiff, E. G.; Popp, R. A. *J Virol* 16(6): 1608-1614; 1975.

3192 IMMUNOLOGICAL STUDIES WITH AVIAN RNA TUMOR VIRUSES. (Eng.) Halpern, M. S. (Univ. Southern California Sch. Medicine, Los Angeles, CA); Hunter, E.; Alevy, M. C.; Friis, R. R.; Vogt, P. K. *Adv Biosci* 12: 567-577; 1974.

3193 GENETIC RECOMBINATION AMONG TEMPERATURE-SENSITIVE MUTANTS OF ROUS SARCOMA VIRUS. (Eng.) Wyke, J. A.; Bell, J. G.; Beamand, J. A. In: *Tumor Viruses. Cold Spring Harbor Symposia on Quantitative Biology*. Cold Spring Harbor Lab. (Cold Spring Harbor, NY): Vol 39, pp. 897-905; 1975.

3194 VIRUS-SPECIFIC RNA IN CELLS INFECTED WITH AVIAN RNA TUMOR VIRUSES (Proceedings Abstract). (Eng.) Brugge, J. S. (Pathology Dept., Univ. Colorado Medical Sch., Denver, CO 80220); Purchio, A. F.; Erikson, R. L. *Fed Proc* 35(7): 1736; 1976.

3195 THE CONTROL OF EXPRESSION OF INSULIN RECEPTORS IN UNINFECTED AND ROUS SARCOMA VIRUS-INFECTED CHICK EMBRYO FIBROBLASTS (Proceedings Abstract). (Eng.) Raizada, M. K. (Lady Davis Inst. Medical Res., Montreal, Canada); Perdue, J. F. *Proc Am Assoc Cancer Res* 17: 86; 1976.

3196 TRANSCRIPTION OF INOSINE-CONTAINING RANDOM COPOLYNUCLEOTIDES BY RNA-DIRECTED DNA POLYMERASE (Proceedings Abstract). (Eng.) Weiss, G. B. (Molecular Hematology Br. NHLI, NIH, Bethesda, MD 20014); Krueger, L. J. *Fed Proc* 35(7): 1592; 1976.

3197 SUBVIRAL PARTICLES WITHIN AND ASSOCIATED WITH THE LYMPHOCYSTIS VIRUS OF FISH (Proceedings Abstract). (Eng.) Dolowy, W. C. (VAH, Hines, IL 60141); Koch, E. A.; Spitzer, R. H.; Greenberg, S.; Brown, E. R. *Proc Am Assoc Cancer Res* 17: 3; 1976.

3198 STIMULATION OF REVERSE TRANSCRIPTASE ACTIVITY BY NUCLEIC ACID-BINDING PROTEIN ISOLATED FROM TRANSFORMED CELLS (Proceedings Abstract). (Eng.) Lee, S. G. (Molecular Virology Lab., Abbott Laboratories, North Chicago, IL 60064); Hung, P. P. *Fed Proc* 35(7): 1592; 1976.

3199 COMPARISON OF THE tRNAS ASSOCIATED WITH THE 70S RNA OF VARIOUS RNA TUMOR VIRUSES (Proceedings Abstract). (Eng.) Waters, L. C. (Biology Div., Oak Ridge Natl. Lab., U.T.-Oak Ridge Graduate Sch. Biomedical Sciences, Oak Ridge, TN 37830); Mullin, B. C. *Fed Proc* 35(7): 1736; 1976.

3200 LYMPHOMA IN *ESOX LUCIUS* (NORTHERN PIKE): VIRAL AND ENVIRONMENTAL INTERACTIONS (Proceedings Abstract). (Eng.) Brown, E. R. (Univ. Health Sciences/Chicago Medical Sch., Chicago, IL 60612); Sinclair, T. F.; Keith, L.; Hazdra, J. J.; Callaghan, O. H.; Inch, W. R. *Proc Am Assoc Cancer Res* 17: 2; 1976.

3201 POLYPEPTIDES OF FL-74 STRAIN OF FELINE LEUKEMIA VIRUS (FeLV) (Proceedings Abstract). (Eng.) Manning, J. S. (Univ. California, Davis, CA 95616); Worley, M. B. *Proc Am Assoc Cancer Res* 17: 116; 1976.

3202 METHYLATION OF FELINE LEUKEMIA VIRUS HIGH MOLECULAR WEIGHT SUBUNIT RNA (Proceedings Abstract). (Eng.) Thomason, A. R. (Michigan State Univ., East Lansing, MI 48824); Brian, D. A.; Velicer, L. F.; Rottman, F. M. *Fed Proc* 35(7): 1372; 1976.

3203 INTRACISTERNAL VIRUS PARTICLES IN SD-MS (RAT BONE TUMOR) VIRUS INFECTED MOUSE PROSTATE CELLS GROWN IN VITRO (Proceedings Abstract). (Eng.) Ohtsuki, Y. (Dept. Virology, Univ. Texas System Cancer Center, M. D. Anderson Hosp. and Tumor Inst., Houston, TX 77025); Seman, G.; Bowen, J. M.; Dmochowski, L. *Proc Am Assoc Cancer Res* 17: 110; 1976.

3204 CHROMATIN STRUCTURE: POSSIBLE REGULATORY ROLE IN GENE EXPRESSION (Proceedings Abstract). (Eng.) Herman, T. M. (Oregon State Univ., Corvallis, OR 97330); Shaw, B. R.; Beaudreau, G. S. *Proc Am Assoc Cancer Res* 17: 32; 1976.

3205 FRIEND LEUKEMIA CELLS (FLC): IN VITRO EFFECTS OF BCNU (Proceedings Abstract). (Eng.) Gabelman, N. (Mount Sinai Sch. Medicine, New York, NY 10029); Rosenthal, L.; Bruckner, H. *Proc Am Assoc Cancer Res* 17: 106; 1976.

3206 ABELSON VIRUS PREVENTS LYMPHOBLASTS FROM CIRCULATING (Proceedings Abstract). (Eng.) Siegler, R. (Dept. Path., Drew Postgrad. Medical Sch., Los Angeles, CA 90059). *Proc Am Assoc Cancer Res* 17: 4; 1976.

3207 CONTAMINATION OF RAT TRANSPLANTABLE TUMOR LINES WITH KILHAM RAT VIRUS: EFFECT ON LYMPHOPROLIFERATIVE RESPONSES IN VITRO (Proceedings Abstract). (Eng.) Campbell, D., Jr. (Natl. Cancer Inst., Bethesda, MD 20014); Manders, E.; Oldham, R.; Bonnard, G.; Staal, S.; Salzman, L.; Herberman, R. *Proc Am Assoc Cancer Res* 17: 156; 1976.

3208 THE MODULATION OF H-2 ANTIGENS IN ABELSON VIRUS-TRANSFORMED LYMPHOBLASTS (Proceedings Abstract). (Eng.) Pratt, D. M. M. (Harvard Medical Sch., Boston, MA 02115); Scher, C. D.; Rosenberg, N.; Parkman, R.; Baltimore, D.; Strominger, J. *Fed Proc* 35(7): 1736; 1976.

3209 INVASION OF THE CHORIOALLANTOIC MEMBRANE BY VIRAL-TRANSFORMED 3T3 CELLS (Proceedings Abstract). (Eng.) Scher, C. D. (Sidney Farber Cancer Center and Children's Hosp., Boston, MA 02115); Haudenschild, C.; Klagsbrun, M. *Proc Am Assoc Cancer Res* 17: 77; 1976.

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3295 EVIDENCE FOR ANIMAL ORIGIN OF CELLS AND RESCUED TUMOUR VIRUS FROM A PUTATIVE HUMAN OSTEOSARCOMA CELL LINE. (Eng.) Hlozanek, I. (Inst. Experimental Biology and Genetics, Czechoslovak Acad. Sciences, Prague 6, Czechoslovakia); Altaner, C.; Donner, L.; Kucerova, M.; Mach, O.; Machala, O.; Sainerova, H.; Sovova, V.; Svoboda, J. *Folia Biol (Praha)* 21(6): 400-405; 1975.

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3297 A COMPARATIVE STUDY OF THE SYNTHESIS AND UTILIZATION OF N^5-METHYL TETRAHYDROFOLATE IN NORMAL AND SV40 TRANSFORMED BHK-21 CELLS (Proceedings Abstract). (Eng.) Jacobsen, S. J. (Brigham Young Univ., Provo, UT 84602); North, J. A.; Rao, N. A. *Fed Proc* 35(7): 1480; 1976.

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3299 THREE NEW VIRUS-CODED LOW MOLECULAR WEIGHT RNA'S IN ADENOVIRUS TYPE 2 INFECTED CELLS (Proceedings Abstract). (Eng.) Varrichio, F. (Memorial Sloan-Kettering Cancer Center, New York, NY 10021); Sehulster, L. M.; Raska, K., Jr. *Fed Proc* 35(7): 1688; 1976.

3300 DEPLETION OF MICROTUBULE-ASSOCIATED-PROTEIN (MAP) CONTENT ABOLISHES SPECIFICITY OF IN VITRO BINDING OF ADENOVIRUS TO MICROTUBULES (Proceedings Abstract). (Eng.) Weatherbee, J. A. (Worcester Foundation Experimental Biology, Shrewsbury, MA 01545); Luftig, R. B.; Weihing, R. R. *Fed Proc* 35(7): 1482; 1976.

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3304 ISOLATION AND CHARACTERIZATION OF AN ENDONUCLEASE FROM ADENOVIRUS TYPE 2-INFECTED KB CELLS (Proceedings Abstract). (Eng.) Reif, U. (Inst. Genetics, Univ. Cologne, Cologne, W. Germany); Winterhoff, U.; Doerfler, W. *Fed Proc* 35(7): 1610; 1976.

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3306 NUCLEOTIDE SEQUENCE HOMOLOGY BETWEEN A VIRUS-SPECIFIC 6S RNA AND VARIOUS RNA SPECIES FROM ADENOVIRUS-2-INFECTED KB CELLS. (Eng.) Ohe, K. (Hiroshima Univ. Sch. Medicine, Kasumi 1-2-3, Hiroshima 734 Japan). *Gann* 66(4): 375-384; 1975.

3307 PURIFICATION AND DNA BINDING OF ADENOVIRUS TYPE 12 TUMOR ANTIGEN (Proceedings Abstract). (Eng.) Raska, K., Jr. (CMDNJ-Rutgers Medical Sch., Piscataway, NJ 08854); Biron, K. K.; Rubio, V. *Proc Am Assoc Cancer Res* 17: 45; 1976.

3308 TRANSCRIPTION MAPS OF ADENOVIRUS TYPE 12 DNA IN INFECTED AND TRANSFORMED CELLS (Abstract). (Eng.) Scheidtmann, K. H. (Institut fuer Genetik der Universitaet zu Koeln, Weyertal 121, D-5000 Koeln 41, W. Germany); Ortin, J.; Greenberg, R.; Westphal, M.; Doerfler, W. *Hoppe Seylers Z Physiol Chem* 357(3): 333; 1976.

3309 INDUCTION OF ONCORNAVIRUS ANTIGENS BY HERPES SIMPLEX VIRUS (Proceedings Abstract). (Eng.) Reed, C. (Pennsylvania State Univ. College Medicine, Hershey, PA 17033); Rapp, F. *Proc Am Assoc Cancer Res* 17: 64; 1976.

3310 RESPONSE TO EPSTEIN-BARR VIRUS IN MALARIA-INFECTED MICE (Proceedings Abstract). (Eng.) Hargis, B. J. (Sidney Farber Cancer Center, Dept. Pathology, Harvard Medical Sch., Boston, MA 02115); Malkiel, S. *Fed Proc* 35(3): 416; 1976.

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3312 DEOXYTHYMIDINE 5'-PHOSPHOROPHOSPHONOACETATE: SYNTHESIS AND EFFECT ON HERPESVIRUS-INDUCED DNA POLYMERASE (Proceedings Abstract). (Eng.) Leinbach, S. S. (Dept. Biochemistry, Michigan State Univ., East Lansing, MI 48823); Lee, L. F.; Boezi, J. A. *Fed Proc* 35(7): 1590; 1976.

3313 ESTABLISHMENT AND CHARACTERIZATION OF AN EPSTEIN-BARR VIRUS (EBV)-NEGATIVE LYMPHOBLASTOID B CELL LINE (BJA-B) FROM AN EXCEPTIONAL, EBV-GENOME-NEGATIVE AFRICAN BURKITT'S LYMPHOMA. (Eng.) Menezes, J. (Dept. Microbiology & Immunology and Pediatrics, Univ. Montreal, Hopital Ste-Justine, 3175, Chemin Cote Ste-Catherine, Montreal, P.Q., Canada); Leibold, W.; Klein, G.; Clements, G. *Biomedicine* 22(4): 276-284; 1975.

3314 CHLAMYDIAL INFECTION IN WOMEN WITH CERVICAL DYSPLASIA. (Eng.) Schachter, J. (George Williams Hooper Foundation, Univ. California, San Francisco, CA 94143); Hill, E. C.; King, E. B.; Coleman, V. R.; Jones, P.; Meyer, K. F. *Am J Obstet Gynecol* 123(1): 753-757; 1975.

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3316 ANTIBODIES TO HERPESVIRUS TYPE 2 IN CARCINOMA OF THE CERVIX UTERI IN IBADAN, NIGERIA. (Eng.) Adelusi, B. (University Coll. Hosp., Ibadan, Nigeria); Osunkoya, B. O.; Fabiyi, A. *Am J Obstet Gynecol* 123(1): 758-761; 1975.

3317 STUDIES WITH HUMAN PAPILLOMA VIRUS DNA (Proceedings Abstract). (Eng.) DeLap, R. (NYU Sch. Medicine, Dept. Biochemistry, 550 First Ave., NY 10016); Yanagi, K.; Rush, M. *Fed Proc* 35(7): 1688; 1976.

3318 ISOLATION OF VIRAL-LIKE PARTICLES FROM HUMAN LEUKEMIC CELLS (Proceedings Abstract). (Eng.) Trousil, E. B. (Dept. Developmental Therapeutics, Univ. Texas System Cancer Center, M. D. Anderson Hosp. and Tumor Inst., Houston, TX 77030); Sahasrabuddhe, C. G.; Saunders, G. F. *Fed Proc* 35(7): 1612; 1976.

3319 SPECIFICITY OF RNA TUMOR VIRUS PROTEIN PHOSPHORYLATION (Proceedings Abstract). (Eng.) Hayman, E. G. (Depts. Pathology and Biochemistry, USC Sch. Medicine, Los Angeles, CA 90033); Roy-Burman, P. *Fed Proc* 35(7): 1610; 1976.

3320 EXAMINATION OF SPLEENS FROM CHRONIC MYELOGENOUS LEUKEMIA (CML) FOR ONCORNAVIRUSES (Proceedings Abstract). (Eng.) Fuscaldo, A. (Cancer Inst., Hahnemann Medical Coll., Philadelphia, PA 19102); Erlick, B.; Bacak, J.; Lamping, C. *Proc Am Assoc Cancer Res* 17: 127; 1976.

3321 POLYOMA VIRUS-INDUCED DEOXYTHYMIDYLATE SYNTHETASE IN BALB-C 3T3 AND BHK-21 CELL LINES (Proceedings Abstract). (Eng.) Amini, R. (Section Biochemistry and Biophysics Univ. Connecticut, Storrs, CT 06268); Roth, J. S. *Fed Proc* 35(7): 1492; 1976.

3322 R-TYPE VIRUS PARTICLES IN PRIMARY HUMAN BRAIN TUMOR CELL CULTURES ASSOCIATED WITH DEFINED CHROMOSOME LOSS AND MEMBRANE ANTIGENS (Proceedings Abstract). (Eng.) Hollinshead, A. (George Washington Univ. Medical Center, Washington, DC 20037); Suskind, G.; Jacobson, C.; Randall, J. *Proc Am Assoc Cancer Res* 17: 211; 1976.

3323 VIRUS-LIKE PARTICLES IN THE SPERM OF NORMAL MEN (Proceedings Abstract). (Eng.) Witkin, S. S. (Lab. Cell Biochemistry, Memorial Sloan-Kettering Cancer Center, New York, NY 10021); Bendich, A. *Proc Am Assoc Cancer Res* 17: 78; 1976.

3324 ANALYSIS OF ONCORNAVIRUS IN HUMAN AND MURINE PLATELETS (Proceedings Abstract). (Eng.) Brodsky, I. (Cancer Inst., Hahnemann Medical Coll., Philadelphia, PA 19102); West, W. *Proc Am Assoc Cancer Res* 17: 127; 1976.

3325 ANTIBODY TO C-TYPE RNA VIRAL PROTEINS IN HUMAN SERA (Proceedings Abstract). (Eng.) Vosika, G. J. (Univ. Minnesota Health Sciences Center, Minneapolis, MN 55455); Balazs, P.; Kennedy, B. J. *Proc Am Assoc Cancer Res* 17: 177; 1976.

3326 CELL SURFACE OF NORMAL AND VIRUS-TRANSFORMED HUMAN CELLS (Proceedings Abstract). (Eng.) Birckbichler, P. J. (Biomedical Div., Noble Foundation, Ardmore, OK 73401); Orr, G. R.; Conway, E.; Carter, H. A.; Patterson, M. K., Jr. *Fed Proc* 35(7): 1682; 1976.

3327 THE CHROMATIN-LIKE STRUCTURE OF THE ADENOVIRUS CHROMOSOME. (Meeting Abstract). (Eng.) Corden, J. (Dept. Biochemistry and Biophysics, Oregon State Univ., Corvallis, OR 97331); Engelking, M.; Pearson, G. *Biophys J* 16(21-part 2): 92a; 1976.

3328 LYMPHOMA INDUCED BY HERPESVIRUS: RESISTANCE ASSOCIATED WITH A MAJOR HISTOCOMPATIBILITY GENE (Proceedings Abstract). (Eng.) Longenecker, B. M. (Depts. Immunology and Zoology, Univ. Alberta, Edmonton, Alberta, Canada); Pazderka, F.; Gavora, J. S.; Ruth, R. F. *Fed Proc* 35(3): 827; 1976.

See also:

* (Rev): 3001, 3024, 3025, 3026, 3027, 3028, 3029, 3053, 3054
* (Phys): 3146, 3147, 3148
* (Immun): 3351, 3356, 3362, 3363, 3366, 3367, 3377, 3383, 3386, 3388, 3389
* (Path): 3421
* (Epid-Biom): 3443

3329 IMMUNOGLOBULINS AND COMPLEMENT RECEPTORS ON CLL CELLS. (Eng.) Grey, H. M. (Natl. Jewish Hosp. and Res. Center, Denver, CO 80206); Kubo, R. T.; Rabellino, E. M.; Polley, M.; Ross, G. D. *Adv Biosci* 12: 213-224; 1974.

Data are presented to support the hypothesis that the predominance of IgM on the surface of chronic lymphatic leukemia (CLL) does not represent a situation found in normal peripheral blood lymphocytes but may be a result of neoplastic transformation or lack of differentiation, or a dedifferentiation, on the part of CLL cells. Two test systems were used to evaluate the relative amounts of IgG- and IgM-bearing cells in normal human peripheral blood: one is an inhibition system using radiolabeled Ig antigens and specific anti-Ig reagents, and the second is a surface iodination and immunoprecipitation procedure using lactoperoxidase to label the lymphocyte surface proteins enzymatically. Both procedures indicated that, in normal human peripheral blood, there is twice as much IgG as IgM associated with the lymphocyte population. The preponderance of the IgM class in CLL does not appear, therefore, representative of normal peripheral blood. The quantitative amounts of Ig on CLL cells also appear to be quite different from what is found on normal lymphocytes. Earlier studies of Ig's associated with CLL cells indicated that about 20% of the cases showed either no Ig or only light chains on the lymphocyte surface. Immunofluorescence with anti-IgD antisera, however, indicated that these were IgD leukemia. These preliminary data suggest that a relatively high incidence of IgD leukemias would be encountered in CLL. In 15/18 CLL cases investigated for complement receptor, leukemic cells reacted preferentially with mouse complement compared to human complement. In six, little or no reactivity with human complement was observed. Further studies indicated that there are two receptor sites for C3 present on normal lymphocytes: one recognized C3b, the only form of C3 on the EAC human; the second recognized a split product of C3b (probably C3d). This latter receptor site would recognize the EAC mouse since both C3b and C3d would be expected to be present on these sensitized erythrocytes. This indicates that in CLL there is a strong tendency for the C3b receptor to be deficient or absent, so that EAC mouse reacts preferentially with these cells. (3 references)

3330 IN VITRO INDUCTION OF CYTOTOXICITY AGAINST SYNGENEIC MASTOCYTOMA AND ITS SUPPRESSION BY SPLEEN AND THYMUS CELLS FROM TUMOR-BEARING MICE. (Eng.) Takei, F. (Dept. Microbiology, Univ. British Columbia, Vancouver, B.C., Canada, V6T 1W5); Levy, J. G.; Kilburn, D. G. *J Immunol* 116(2): 288-293; 1976.

Tumor-specific immunity in DBA/2 mice during the growth of a transplantable syngeneic mastocytoma (P815) was assessed. Immunity was measured by the in vitro generation of cytotoxic cells. P815 x 2 mastocytoma cells injected sc into syngeneic DBA/2 mice, induced T lymphocyte-mediated cytotoxicity in the mice. During tumor growth cytotoxic activity decreased and ultimately the tumor killed the mice. When spleen cells from mice with tumors in the early stages of growth were incubated in vitro with mitomycin C-treated tumor cells, specific cytotoxicity mediated by T lymphocytes was developed. However, spleen cells, taken from mice with tumors at a later growth stage, did not develop cytotoxic activity. The unresponsiveness of spleen cells from mice with tumors in the latter stages of growth seemed to be due to the presence of suppressor cells since in vitro development of cytotoxicity by spleen cells from early tumor-bearing mice were inhibited by the addition of spleen cells or thymocytes from mice with progressively growing tumors. Normal spleen cells or thymocytes did not affect the response.

3331 CHARACTERIZATION OF A TL- VARIANT OF A HOMOZYGOUS TL+ MOUSE LYMPHOMA. (Eng.) Hyman, R. (Salk Inst. Biological Studies, San Diego, CA 92112); Stallings, V. *Immunogenet* 3(1): 75-84; 1976.

A homozygous antigen-loss variant for the TL antigen was isolated by immunoselection in vitro. The variant expressed less than 0.01 of the parental amount of TL antigen on its surface as measured by quantitative absorption. Neither the K nor the D end H-2k antigens were detectable on the surface of the variant, although parental amounts of Thy 1.2 and GCSA were expressed. Karyotypic analysis showed that the variant had one less chromosome than the parental line. It was concluded that TL antigen on the cell surface is not necessary for cell survival.

3332 B AND T CELLS IN VARIOUS DISEASE STATES: IMMATURE B CELLS IN THE BLOOD OF CERTAIN PATIENTS WITH IMMUNE DEFICIENCY. (Eng.) Wernet, P. (Rockefeller Univ., New York, NY 10021); Siegal, F. P.; Dickler, H.; Bentwich, Z.; Fu, S.; Kunkel, H. G. *Adv Biosci* 12: 169-184; 1974.

The nature of the Ig negative, Fc receptor positive, and nonrosetting lymphocyte population found in chronic lymphocytic leukemia (CLL) patients with various types of immunodeficiency states was investigated. Lymphocytes from 11 CLL patients selected partly for weak surface Ig staining were compared for binding of aggregated IgG and surface Ig staining. In 10 that had appreciable numbers of positive lymphocytes, a relative homogeneity of distribution and intensity of staining was present in each case for both aggregate binding and surface Ig. Four either lacked, or showed small numbers of, lymphocytes that stained for surface Ig, but a high proportion of their lymphocytes bound aggregates. Only in one individual were the abnormal cells identified as T-cells, while the bulk of the cases showed B lymphocytes. The presence of the Fc receptor was studied by uptake of aggregated γ-globulin in four patients with immune deficiency. In two, the Fc receptor was clearly present, indicating a significant number of B cells. In the third, no Ig-bearing cells were observed during initial studies. The fourth case showed some Ig-bearing cells in the freshly drawn blood, but the cells proved to be IgG and disappeared after overnight culture. Some cells appeared to represent immature B cells that could be stimulated to form surface Ig by short-term culture; they could also be visualized by enzymatic surface radioiodination. Evidence was obtained for a B cell maturation factor that is produced by the thymus cells and is present in normal serum but is absent in the serum of these two patients. Further analysis of B cell development and function by combined studies of the Ig characteristics of peripheral blood lymphocytes and the in vitro synthesis of Ig may aid in the understanding of these disorders. (16 references)

3333 IMMUNODEFICIENCY AND MALIGNANCY. (Eng.)

Good, R. A. In: *Host Environment Interactions in the Etiology of Cancer in Man. Proceedings of a Meeting Held at Primosten, Yugoslavia, 27 August-2 September 1972.* International Agency for Res. on Cancer and John E. Fogarty International Center of the Natl. Insts. of Health USA. (Lyon, France): pp. 265-274; 1973.

The relation between immunodeficiency and malignancy is discussed, with use of data from the literature and previously unpublished data. Multiple myeloma, Hodgkin's disease, chronic lymphatic leukemia, acute lymphatic leukemia, and acute myelocytic leukemia are all associated with secondary immunodeficiencies. Several immunodeficiency diseases in which malignancy appears in higher than expected numbers, implying that it is secondary to the disease, are also discussed. Bruton-type X-linked infantile agammaglobulinemia, a disease characterized by deficiency or absence of IgM, IgA, IgG, IgD, and IgE, was associated with a 5%-10% incidence of cancer (70 cases of leukemia, 6 fatal, and 1 brain tumor). Ataxia-telangiectasia, which is characteristically associated with T-cell, B-cell, IgA, and IgE deficiency, was associated with a 10%-15% incidence of many different forms of cancer (47 cases). The Wiskott-Aldrich syndrome, which is characterized by low levels of IgM, was associated with a greater than 10% incidence of cancer (17 cases), mostly lymphoreticular malignancies. Other immunodeficiency diseases, such as the DiGeorge syndrome and reticular dysgenesis, are also discussed.

3334 CYCLIC GMP AND LECTIN-INDUCED LYMPHOCYTE ACTIVATION. (Eng.)

Wedner, H. J. (Washington Univ. Sch. Medicine, St. Louis, MO 63110); Dankner, R.; Parker, C. W. *J Immunol* 115(6): 1682-1687; 1975.

Purified human peripheral lymphocytes (incubated with phytohemagglutinin and concanavalin A) were examined for alterations in intracellular cyclic guanosine monophosphate (cGMP) and cyclic adenosine monophosphate (cAMP) using multiple techniques for the isolation and purification of cGMP and cAMP before assay of the cyclic nucleotides by radioimmunoassay. Consistent increases in cGMP (under any of the experimental conditions used and with any of the various purification schemes) were not demonstrated. In these same experiments, exogenous cGMP added to the lymphocytes could be measured, and the immunoreactive material was destroyed by cyclic nucleotide phosphodiesterase, which indicated that the inability to measure increases in cGMP was not caused by the inability to measure cGMP. Under identical experimental conditions, small (but consistent and statistically significant) increases in cAMP were noted. Other parameters of lymphocyte activation, ^{45}Ca uptake (an early parameter), and incorporation of ^{3}H-thymidine into DNA were unimpaired. With this data, the concept of cGMP as the second messenger in lectin-stimulated human peripheral lymphocytes became questionable.

3335 DECREASE AND ALTERED DISTRIBUTION OF HUMAN T ANTIGEN ON CHRONIC LYMPHATIC CELLS OF T TYPE, SUGGESTING A CLONAL ORIGIN. (Eng.)

Thiel, E. (8 Muenchen 2, Landwehrstr. 61, Institut fuer Haematologie, W. Germany); Rodt, H.; Huhn, D.; Thierfelder, S. *Blood* 47(5): 723-726; 1976.

B and T cell markers were studied by immunoautoradiography in a patient with chronic lymphocytic leukemia and erythroderma, and in a normal subject. The absence of immunoglobulin, complement receptor, and Fc receptor, and the presence of SRBC receptor and T cell antigen on the membrane of the leukemic cells classified them as T cells. By quantitative microphotometric immunoautoradiography, surface antigen densities were measured at the cellular level with the following results: (a) the density of the T antigen sites was less on leukemic cells compared to normal T lymphocytes. (b) The T antigen densities of leukemic lymphocytes varied less from cell to cell, forming a homogeneous peak in histograms. (c) Ig density of normal B lymphocytes was demonstrated on the residual T antigen-negative cells. The results were confirmed qualitatively by direct immunofluorescence and electron microscopy with peroxidase-labeled antibodies. The surface antigens were also quantitatively measured at the cell population level by a quantitative microcomplement fixation test that revealed reduced binding of anti-T cell antibodies and complement, compared to the normal lymphocytes and no antiglobulin fixation on the leukemic lymphocytes. As lymphocytes with normal T antigen concentration could not be found among the leukemic T lymphocytes, a lack of normal T cells was assumed. The findings that there was a decrease and altered distribution of surface markers on chronic lymphatic leukemia cells of the B and T cell type augment the argument that these cells are of clonal origin in thymus-derived chronic lymphatic leukemia.

3336 THE EFFECT OF ALLOANTISERA ON ANTIGEN-INDUCED T CELL PROLIFERATION. (Eng.)

Ruhl, H. (Natl. Inst. Allergy and Infectious Diseases, Natl. Inst. Health, Bethesda, MD 20014); Shevach*, E. M. *J Immunol* 115(6): 1493-1499; 1975.

In the guinea pig, alloantisera raised by cross-immunization of strain 2 and strain 13 animals were capable of specifically inhibiting the in vitro proliferative response of $(2 \times 13)F_1$ T lymphocytes to those antigens the response to which was controlled by immune response genes (linked to the genes controlling the alloantigens against which the serum was directed). In similar studies performed in the two parental strains, the responses to antigens not known to be under unigenic control were also markedly inhibited by the appropriate alloantisera. In an extension of these studies, the proliferative response of strain 2 and strain 13 T cells to a large number of antigens was markedly inhibited by anti-2 and anti-13 sera. Antisera to the B alloantigen, the product of a linked but distinct histocompatibility locus, which was present in both strain 2 and strain 13 animals, also produced a marked inhibition of T-lymphocyte proliferation.

3337 THE COMPARATIVE SPECIFICITY OF THREE OESTRADIOL-BINDING PROTEINS: RAT α-FOETOPROTEIN, RAT LIVER 17β-HYDROXY STEROID DEHYDROGENASE AND ANTI-(OESTRADIOL-6-CARBOXYMETHYLOXIME--BOVINE SERUM ALBUMIN) ANTISERUM. (Eng.) Laurent, C. (Laboratoire Associe 87 du CNRS, UER Biomedicale des Saints-Peres, 45 rue des Saints-Peres, 75270-Paris Cedex 06, France); de Lauzon, S.; Cittanova, N.; Nunez, E.; Jayle, M.-F. *Biochem J* 151(13): 513-518; 1975.

A study was made of the specificity of 3-estradiol-binding proteins. Two of these proteins were naturally occurring (rat α-fetoprotein and rat liver microsomal 17β-hydroxy steroid dehydrogenase); the third was an artifically induced model, anti-(estradiol-6-carboxymethyloxime-bovine serum albumin) γ-globulins. A specific binding procedure for each protein model permitted a determination of its affinity for estradiol and 30 other steroids. The results obtained brought to light the different areas of the steroid molecule important for its recognition by each of the three proteins. The two naturally occurring proteins (α-fetoprotein and 17β-hydroxy steroid dehydrogenase) recognized the edge of the steroid defined by C-4, C-6, C-8 and C-15. The γ-globulins recognized the opposite edge. Diethylstilbestrol was only recognized by the two naturally occurring proteins.

3338 ACTIVATION IN VITRO OF MOUSE MACROPHAGES BY SYNGENEIC, ALLOGENEIC, OR XENOGENEIC LYMPHOCYTE SUPERNATANTS. (Eng.) Fidler, I. J. (Frederick Cancer Res. Center, P.O. Box B, Frederick, MD 21701). *J Natl Cancer Inst* 55(5): 1159-1163; 1975.

The macrophages from normal C57BL/6 mice, those with a sc B16 melanoma, and mice immunized against the tumor were examined for in vitro cytotoxicity to B16 tumor cells. Macrophages were treated by incubation with supernatants from B16 cells grown either in unmixed cultures or in cultures containing syngeneic, normal, or sensitized allogeneic (A mouse), or xenogeneic (rat) lymphocytes. The various treated and untreated macrophages were then cultured for five days with viable B16 cells prelabeled with ^{125}I-5-iodo-2'-deoxyuridine; the cultures were terminated, and the extent of destruction of the B16 target cells was determined from the amounts of radioactivity remaining in adherent tumor cells. Untreated macrophages from immunized mice were cytotoxic to the tumor cells; macrophages from normal and tumor-bearing mice became cytotoxic by incubation with supernatants from cultures containing lymphocytes from immunized syngeneic mice, sensitized allogeneic mice, or sensitized rats; macrophages incubated with supernatants from cultures containing normal nonsensitized allogeneic or xenogeneic lymphocytes showed no cytotoxicity. Macrophages from tumor-bearing animals were thought to be potentially cytotoxic to their syngeneic tumors.

3339 HYBRID-VERSUS-PARENTAL STRAIN REACTION; I. COMPARATIVE MORTALITY IN ALLOGENEIC AND PARENTAL STRAIN MALE AND FEMALE MICE. (Eng.) Uphoff, D. E. (Natl. Cancer Inst., Bethesda, MD 20014). *J Natl Cancer Inst* 55(5): 1213-1215; 1975.

The lethally irradiated male and female hybrids, parental strains, and the third party strain A mice were inoculated with reciprocal hybrid marrow from donors of the same sex. A graft-versus-host reaction was produced against H-Y antigens of reciprocal hybrids. Although the late mortality in inbred strain males was generally greater than that of females, this difference was not automatically attributable to an X or a Y chromosome disparity. Hybrids did not recognize the qualitative difference in antigenicity of their reciprocals. However, they were able to recognize the quantitatively greater antigenicity of their parental strains, and a hybrid-versus-parental strain reaction occurred. The graft-versus-host reaction in the third party strain A mice was more severe than that produced by hybrid marrow in their reciprocal hybrid recipients (but less severe than that produced in the parental strains). The severity of the graft-versus-host reactions masked the maternal influences observed with other reciprocal hybrids.

3340 ABILITY OF LYMPHOID CELLS FROM SENSITIZED ALLOGENEIC DONORS TO TRANSFER TUMOUR RESISTANCE. (Eng.) Jurin, M.; Suit, H. D. In: *Host Environment Interactions in the Etiology of Cancer in Man. Proceedings of a Meeting Held at Primosten, Yugoslavia, 27 August-2 September 1972.* International Agency for Res. on Cancer. (Lyon, France): pp. 427-433; 1973.

The factors that determine whether resistance can be transferred from specifically sensitized allogeneic and syngeneic donors and the duration of this resistance were investigated. Two tumors, a methylcholanthrene-induced fibrosarcoma and an LP59 cell tumor (both syngeneic to C3Hf/Bu mice), were assayed with C57BL/6 mice as allogeneic donors. Following heavy irradiation (10,000 rads), three injections of 2 x 10^7 fibrosarcoma, LP59 tumor, or lymphoid cells were given at 7-day intervals. One week after the last dose, some mice were killed and a lymphoid cell suspension was prepared. To transfer immunity, each mouse received two iv injections of 10^8 viable lymphoid cells at 2-day intervals. Three categories of C3Hf/Bu recipients resulted: untreated, tumor-immunized, and those injected with donor lymphoid cells from donors of various categories. Viable tumor cells were injected into the right flank of 5-20 mice for each cell dose tested. At 120 days post-transplantation, the transplant-take results for each assay were tabulated and a logit regression line was fitted through the data. From this line, the TD_{50} dose was calculated. Resistance transfer against the fibrosarcoma from specifically sensitized allogeneic or syngeneic donors was not successful; even 2 x 10^8 viable lymphoid cells had no effect. Resistance to the LP59 tumor was transferred by means of two injections of 10^8 lymphoid cells but not by a single injection of 5 x 10^7 cells from specifically immunized allogeneic or syngeneic donors. After three injections of radiation-killed tumor cells, the TD_{50} was higher by a factor of 100 for the LP59 tumor but only by a factor of 20 for the fibrosarcoma. Transferred resistance to LP59 was maintained for at least 6 wk after injection of lymphoid cells; this cannot be due to the direct action of donor cells because such cells disappear from the host soon after injection. It was concluded that the antigenicity of the tumor and the quantity of injected lymphoid cells from a sensitized donor are the decisive factors in the induction and expression of tumor resistance. (28 references)

3341 SPECIFIC POTENTIATION OF L1210 VACCINE BY PYRAN COPOLYMER. (Eng.) Mohr, S. J. (Natl. Cancer Inst., Natl. Insts. Health, Bethesda, MD 20014); Chirigos, M. A.; Smith, G. T.; Fuhrman, F. S. *Cancer Res* 36(6): 2035-2039; 1976.

To demonstrate the adjuvant capability of the pyran copolymer (NSC 46015), pyran was combined with a vaccine prepared by radiation of L1210 ascites cells and used in a suboptimal manner. It was found to potentiate strongly the immune response of C57BL/6J x DBA/2 F_1 mice to 10^4 live L1210 tumor cells following suboptimal vaccination with 10^7 radiation-inactivated L1210 cells. Optimal immunity to challenge was produced by concomitant ip administration of pyran and L1210 vaccine, and activity was dependent upon both pyran and vaccine dosages. Neither agent alone enhanced this immunity. This immunopotentiation also appeared related to the intrinsic viscosity of different preparations tested, although all the pyran compounds had significant activity. The activity of pyran appeared related to the molecular wt of the polymer; it also had a widely effective dosage range. The range for the vaccine was limited. Furthermore, the increased immunity to subsequent live tumor challenge appeared to be specific for the vaccinating cell type.

3342 ADAPTATION TO HUMAN AND ANIMAL ORGAN ALLOGRAFTS: RECONSIDERATION OF A HYPOTHESIS. (Eng.) Miller, J. (Veterans Admin. Hosp., Minneapolis, MN 55417); Lifton, J.; DeWolf, W. C.; Hattler, B. G., Jr. *Transplant Proc* 8(2): 217-224; 1976.

The clinical results at a center for renal transplants show that patients who underwent preoperative splenectomy had no detectable serum mixed lymphocyte reaction (MLR) after at least 24 mo of followup. Five patients who did not undergo splenectomy all had detectable IgG blocking factors 12 mo posttransplant that caused MLR inhibition of cultures of donor responding cells. Some experimental studies were then done to see if shedding of the determinants that stimulate MLR observed in vitro could be reproduced and accelerated in transplant experiments. Inbred mouse strains BALB/c and DBA/2 underwent skin allografts and received pretreatment with splenic lymphocytes or phytohemagglutinin for 2 wk. A significant prolongation of BALB/c skin allograft survival occurred on 8-wk-old DBA/2 recipients pretreated with cultured cells. An in vitro prolongation of canine allograft survival was seen after pretreating the donor kidneys with infusions of concanavalin A (Con A). Canine kidney transplants that were perfused in Ringers solution and Con A provided a significant increase (30%) in the recipient survival. The same dosage of Con A given iv at the time of the transplant had no effect upon survival. An in vitro assay, the lymphocyte kidney reaction (LKR), has been developed to determine antigenic modulation of the kidney allograft. In dogs, when weak MLR reactivity between peripheral blood lymphocytes of the donor and recipient is present, the LKR is also weak, and vice versa. (33 references)

3343 MODIFICATION OF CANCER BY STRESS; EFFECTS OF PLASMA CORTICOSTERONE ELEVATIONS ON IMMUNOLOGICAL SYSTEM COMPONENTS IN MICE (Proceedings Abstract). (Eng.) Spackman, D. (Pacific Northwest Res. Foundation, Seattle, WA 98104); Riley, V. *Fed Proc* 35(7): 1693; 1976.

3344 HUMORAL IMMUNE RESPONSE TO CHEMICALLY INDUCED SARCOMAS (Proceedings Abstract). (Eng.) Parker, G. A. (Surgery Branch, NCI, NIH, Bethesda, MD 20014); Rosenberg, S. A. *Proc Am Assoc Cancer Res* 17: 80; 1976.

3345 DEVELOPMENT OF REDUCED IMMUNOLOGICAL RESPONSIVENESS IN TUMOR-BEARING MICE (Proceedings Abstract). (Eng.) Biddison, W. E. (Wistar Inst., Philadelphia, PA 19104); Palmer, J. C. *Fed Proc* 35(3): 827; 1976.

3346 THE EFFECTS OF AGE ON THE IMMUNE RESPONSE TO TYPE III PNEUMOCOCCAL POLYSACCHARIDE (SIII) AND BACTERIAL LIPOPOLYSACCHARIDE (LPS) IN BALB/C, SJL/J, AND C3H MICE. (Eng.) Smith, A. M. (East Carolina Univ. Sch. of Medicine, Greenville, NC 27834). *J Immunol* 116(2): 469-474; 1976.

3347 CELL-MEDIATED IMMUNE RESPONSE OF PATIENTS WITH MENINGIOMAS DEFINED IN VITRO BY A [^{3}H]PROLINE MICROCYTOTOXICITY TEST. (Eng.) Pees, H. W. (Institut Medizinische Universitaetsklinik, D-665 Homburg/Saar, W. Germany); Seidel, B. *Clin Exp Immunol* 24(2): 310-316; 1976.

3348 IN VITRO IMMUNE RESPONSE TO THE 2,4,6-TRINITROPHENYL DETERMINANT IN AGED C57BL/6J MICE: CHANGES IN THE HUMORAL IMMUNE RESPONSE TO, AVIDITY FOR THE TNP DETERMINANT AND RESPONSIVENESS TO LPS EFFECT WITH AGING. (Eng.) Kishimoto, S. (Second Dept. Internal Medicine, Kumamoto Univ. Medical Sch., Kumamoto, Japan 860); Takahama, T.; Mizumachi, H. *J Immunol* 116(2): 294-300; 1976.

3349 OXISURAN AND IMMUNE REACTIONS: MEDIATION OF OXISURAN ACTION BY THE ADRENAL GLANDS. (Eng.) van Dijk, H. (Dept. Immunology, State Univ. Utrecht, Catharijnesingel 59, Utrecht, The Netherlands); Bakker, I. A.; Testerink, J.; Bloksma, N.; Willers, J. M. *J Immunol* 115(6): 1587-1591; 1975.

3350 RESISTANCE AND CELL MEDIATED IMMUNITY TO SPONTANEOUS LYMPHOMA CELLS OF AKR/J MICE BY AKR/CU MICE (Proceedings Abstract). (Eng.) Zatz, M. M. (NIH, Bethesda, MD 20014). *Fed Proc* 35(3): 827; 1976.

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3352 MACROPHAGES AND ANTITUMOR IMMUNITY (Proceedings Abstract). (Fre.) Simar, L. J. (Laboratoire d'Anatomie Pathologique, Univ. Liege, 1, rue des Bonnes-Villes, B-4000 Liege, Belgium); Boniver, J.; Courtoy, R. *J Microsc Biol Cell* 23(2): 78a; 1975.

3353 INHIBITION OF THE IN VITRO 19S AND 7S ANTIBODY RESPONSE BY IMMUNOGLOBULIN-BINDING FACTOR (IBF) FROM ALLOANTIGEN-ACTIVATED T CELLS. (Eng.) Gisler, R. H. (Pharmaceuticals Div., Ciba-Geigy Ltd., 4002-Basel, Switzerland); Fridman, W. H. *Cell Immunol* 23(1): 99-107; 1976.

3354 SUPPRESSED HETEROGENEOUS ANTINUCLEAR ANTIBODY RESPONSE IN LYMPHOMA-BEARING NZB/NZW MICE. (Eng.) Walker, S. E. (R4633 Kresge Building, Univ. Michigan Medical Center, Ann Arbor, MI 48104); Bole, G. G., Jr. *Clin Exp Immunol* 24(1): 210-217; 1976.

3355 IMMUNOLOGICAL CROSS-REACTIVITY OF ANTIBODIES TO A SYNTHETIC UNDECAPEPTIDE OF CARCINOEMBRYONIC ANTIGEN WITH THE INTACT PROTEIN AND WITH HUMAN SERA (Abstract). (Eng.) Arnon, R. (Dept. Chemical Immunology, Weizmann Inst. Science, Rehovot, Israel); Bustin, M.; Calef, E.; Chaichik, S.; Haimovich, J.; Novik, N.; Sela, M. *Israel J Med Sci* 11(12): 1399; 1975.

3356 ANTIVIRAL AND ANTITUMOR ANTIBODIES IN MOUSE AND HUMAN BREAST CANCER (Proceedings Abstract). (Eng.) Bowen, J. M. (Dept. Virology, Univ. Texas System Cancer Center, M. D. Anderson Hosp. and Tumor Inst., Houston, TX 77025); Dmochowski, L.; Seman, G.; Scanlon, M. *Proc Am Assoc Cancer Res* 17: 58; 1976.

3357 STRUCTURAL IDENTITY OF A MURINE MYELOMA WITH AN INDUCED HOMOGENEOUS ANTIBODY (Proceedings Abstract). (Eng.) Meyers, G. L. (Roswell Park Memorial Inst., Buffalo, NY 14263); Bankert, R. B.; Pressman, D. *Fed Proc* 35(7): 1650; 1976.

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3359 STUDIES ON THE ATTACHMENT OF SENSITIZED SHEEP ERYTHROCYTES TO TUMORS (Proceedings Abstract). (Eng.) Targowski, S. (Dept. Microbiology, Sch. Medicine, SUNY, Buffalo, NY 14214); Abeyounis, C. J.; Milgrom, F. *Fed Proc* 35(3): 1887; 1976.

3360 ISOLATION AND CHARACTERIZATION OF HUMAN LEUKEMIA-ASSOCIATED ANTIGENS FROM A CHRONIC MYELOGENOUS LEUKEMIA CELL LINE K-562 (Proceedings Abstract). (Eng.) Collins, J. L. (Univ. Tennessee, Knoxville, TN 37916); Wust, C. J.; Lozzio, B. B.; Lozzio, C. B. *Fed Proc* 35(3): 413; 1976.

3361 CHICKEN FETAL ANTIGEN: ITS PHYLOGENETIC DISTRIBUTION AND GENETIC REGULATION (Proceedings Abstract). (Eng.) Dietert, R. R. (Dept. Zoology, Univ. Texas, Austin, TX 78712); Sanders, B. G. *Fed Proc* 35(3): 413; 1976.

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3364 CHANGE OF ANTIGENIC EXPRESSION ON RAT TUMOR CELLS AFTER THEIR TRANSPLANTATION. (Eng.) Gotohda, E. (Hokkaido Univ. Sch. Medicine, Sapporo, Japan); Sendo, F.; Nakayama, M.; Hosokawa, M.; Kawamura, T.; Kodama, T.; Kobayashi, H. *J Natl Cancer Inst* 55(5): 1079-1083; 1975.

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3366 CONCERNING AN EXTRACELLULAR PARTICULATE FROM A CONTINUOUS CULTURE (J111) DERIVED FROM HUMAN LEUKEMIA (Proceedings Abstract). (Eng.) Smith, C. C. (Dept. Epidemiology, Univ. Michigan, Ann Arbor, MI 48109); Dabich, L.; Maverakis, N. H.; Ackermann, W. W. *Fed Proc* 35(3): 548; 1976.

3367 PHYSICAL PROPERTIES OF TWO IMMUNOLOGICALLY DISTINCT PLASMINOGEN ACTIVATORS FROM HAMSTER CELL LINES (Proceedings Abstract). (Eng.) Christman, J. K. (Mt. Sinai Sch. Medicine, New York, NY 10029); Whalen, C.; Silverstein, S. C.; Acs, G. *Fed Proc* 35(7): 1462; 1976.

3368 COMPARISON BETWEEN HUMAN AND MURINE LYMPHOCYTE MEMBRANE IMMUNOGLOBULINS (Proceedings Abstract). (Eng.) Yamaga, K. (National Jewish Hosp. and Res. Center, Denver, CO 80206); Kubo, R. T. *Fed Proc* 35(3): 1150; 1976.

3369 HUMAN MYELOMA IgA HALF-MOLECULES (Proceedings ABSTRACT). (Eng.) Fishkin, B. G. (Wadsworth VAC and UCLA, Los Angeles, CA 90073); Speigelberg, H. L. *Fed Proc* 35(3): 313; 1976.

3370 STRUCTURAL STUDIES OF IgD (Proceedings Abstract). (Eng.) Goyert, S. M. (Scripps Clinic and Res. Foundation, La Jolla, CA 92037); Hugli, T. E.; Meinke, G. C.; Spiegelberg, H. L. *Fed Proc* 35(3): 313; 1976.

3371 IgE-IgM KAPPA GAMMOPATHY ASSOCIATED WITH LYMPHOCYTIC LYMPHOMA (Proceedings Abstract). (Eng.) Brown, G. L. (Fitzsimons Army Medical Center, Denver, CO 80240); DiBella, N. J.; Corby, D. G. *Fed Proc* 35(3): 438; 1976.

3372 THE CLEAVAGE SITES OF HUMAN IgG1 MYELOMA PROTEINS BY PAPAIN (Proceedings Abstract). (Eng.) Wang, A.-C. (Medical Univ. South Carolina, Charleston, SC 29401); Wang, I. Y. *Fed Proc* 35(3): 313; 1976.

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3374 FACTORS AFFECTING THE TRANSLATION OF IMMUNOGLOBULIN mRNA IN CELL-FREE SYSTEMS (Proceedings Abstract). (Eng.) Elahi, E. (Univ. Michigan, Ann Arbor, MI 48109); Jones, G. H. *Fed Proc* 35(7): 1439; 1976.

3375 THE IMMUNOHISTOLOGIC LOCALIZATION OF IMMUNOGLOBULINS AND COMPLEMENT IN NORMAL, BENIGN AND MALIGNANT HUMAN BREAST TISSUE (Proceedings Abstract). (Eng.) Richman, A. V. (Univ. Florida Coll. Medicine, Gainesville, FL 32610). *Proc Am Assoc Cancer Res* 17: 57; 1976.

3376 IDENTIFICATION OF HEAVY CHAIN TYROSINE 33 IN THE BINDING SITE OF MYELOMA PROTEIN MCPC 603 BY PAIRED LABEL IODINATION (Proceedings Abstract). (Eng.) Grossberg, A. L. (Roswell Park Memorial Inst., Buffalo, NY 14263); Roholt, O. A.; Pressman, D. *Fed Proc* 35(3): 273; 1976.

3377 MITOGENICITY OF THROMBIN FOR NORMAL AND TRANSFORMED CHICK EMBRYO FIBROBLASTS (Proceedings Abstract). (Eng.) Chen, L. B. (Dept. Biology, Massachusetts Inst. Technology, Cambridge, MA 02139); Zetter, B. R. *Fed Proc* 35(7): 1462; 1976.

3378 ROSETTE FORMATION WITH MOUSE ERYTHROCYTES: PROBABLE MARKER FOR HUMAN B LYMPHOCYTES. (Eng.) Gupta, S. (Roosevelt Hosp., 428 West 59th St., New York, NY 10019); Grieco, M. H. *Int Arch Allergy Appl Immunol* 49(6): 734-742; 1975.

3379 IMMUNOLOGIC FUNCTIONS OF ISOLATED HUMAN LYMPHOCYTE SUBPOPULATIONS: VI; FURTHER CHARACTERIZATION OF THE SURFACE Ig NEGATIVE, E ROSETTE NEGATIVE (NULL CELL) SUBSET. (Eng.) Chess, L. (Sidney Farber Cancer Center, Boston, MA 02115); Levine, H.; MacDermott, R. P.; Schlossman, S. F. *J Immunol* 115(6): 1483-1487; 1975.

3380 CONTACTS BETWEEN CYTOTOXIC LYMPHOID CELLS AND TUMOR TARGET CELLS. TRANSMISSION AND SCANNING ELECTRON MICROSCOPIC STUDY (Proceedings Abstract). (Fre.) Firket, H. (Laboratoire d'Anatomie Pathologique, Universite de Liege, 1, rue des Bonnes-Villes, B-4000 Liege, Belgium); Degiovannni, G.; Lafontaine, N. *J Microsc Biol Cell* 23(2): 47a; 1975.

3381 AUGMENTATION OF PROLIFERATION AND IN VITRO PRODUCTION OF CYTOTOXIC CELLS BY 2-ME IN THE RAT. (Eng.) Soulillou, J.-P. (Peter Bent Brigham Hosp., Boston, MA 02115); Carpenter, C. B.; Lundin, A. P.; Strom, T. B. *J Immunol* 115(6): 1566-1571; 1975.

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3384 IMMUNOLOGICAL SIDE-EFFECTS OF ANTICONVULSANTS (Letter to Editor). (Eng.) Massimo, L. (Dept. Pediatric Hematology and Oncology, Istituto G. Gaslini, Genoa, Italy); Pasino, M,; Rosanda-Vadala, C.; Tonini, G. P.; De Negri, M,; Saccomani, L. *Lancet* 1(7964): 860; 1976.

3385 PREPARATION AND PROPERTIES OF AN ANTIPLASMA CELL SERUM DIRECTED AGAINST A DEFINED PLASMACYTOMA CELL POPULATION. (Eng.) Proctor, M. L. (Georgetown Univ. Sch. Medicine and Dentistry, Washington, DC 20007); Herscowitz*, H. B. *J Immunol* 115(6): 1642-1649; 1975.

3386 IMMUNITY AND SURVIVAL. (Eng.) Buckley, C. E., III (Duke Univ. Medical Center, Durham, NC 22710); Roseman, J. M. *J Am Geriatr Soc* 24(6): 241-248; 1976.

3387 THE ROLE OF IMMUNE FACTORS IN THE SURVIVAL OF CIRCULATING TUMOR CELLS. (Eng.) Wexler, H. (Natl. Cancer Inst., Natl. Insts. Health, Bethesda, MD 20014); Sindelar, W. F.; Ketcham, A. S. *Cancer* 37(4): 1701-1706; 1976.

3388 IMMUNE DEFICIT IN EPIDERMODYSPLASIA VERRUCIFORMIS (Letter to Editor). (Fre.) Lambert, D. (Hopital du Bocage, F 21034 Dijon Cedex, France); Laurent, R.; Turc, J.-M.; Gambert, I.; Gavanou, J.; Chapuis, J.-L. *Nouv Presse Med* 5(11): 719; 1976. (No references)

3389 IMMUNOGENICITY OF VIRUS-INDUCED RAT OSTEOSARCOMA. (Eng.) Urovitz, E. P. (Univ. Toronto, Ontario, M5G 1X5, Canada); Czitrom, A. A.; Langer, F.; Gross, A. E.; Pritzker, K. P. H. *J Bone Joint Surg* 58-A(3): 308-311; 1976.

3390 CHARACTERIZATION OF SMALL LYMPHOCYTES IN THE EHRLICH ASCITES TUMOR (Proceedings Abstract). (Eng.) Lala, P. K. (Dept. Anatomy, McGill Univ., Montreal, Canada); Garnis, S. *Proc Am Assoc Cancer Res* 17: 153; 1976.

3391 POTENTIATION BY CARBON TETRACHLORIDE OF THE IMMUNOSUPPRESSIVE EFFECTS OF FIBROSARCOMA AND TOXIC MATERIAL PRODUCED BY FIBROSARCOMA CELLS. (Eng.) Wong, A. O. B. (Kingston General Hosp., Kingston, Ontario, Canada); Mankovitz, R.; Kennedy*, J. C. *J Natl Cancer Inst* 55(5): 1113-1118; 1975.

See also:

* (Rev): 3003, 3007, 3023, 3025, 3028, 3029, 3030, 3033, 3031, 3032, 3033, 3034, 3035, 3036, 3037, 3054, 3055
* (Phys): 3146, 3148
* (Viral): 3163, 3167, 3183, 3188, 3190, 3206, 3207, 3212, 3218, 3219, 3225, 3226, 3231, 3233, 3234, 3242, 3249, 3252, 3253, 3257, 3267, 3272, 3277, 3293, 3296, 3309, 3310, 3316, 3322, 3325, 3328
* (Epid-Biom): 3443

3392 ABNORMALITIES OF CHROMOSOME 1 IN MYELOPROLIFERATIVE DISORDERS. (Eng.) Rowley, J. D. (Dept. Medicine, Box 420, Univ. Chicago, Chicago, IL 60637). *Cancer* 36(5): 1748-1757; 1975.

A total of three patients with myeloproliferative disorders showed a similar chromosome abnormality (accompanied by other abnormalities that were different in each case). Marrow cells from all three patients were trisomic either for the entire chromosome 1 or for its long arm. Patient 1 had a brief period of anemia and thrombocytopenia preceding a terminal acute leukemia; Patient 2 had polycythemia vera (PV) that terminated in acute leukemia; and Patient 3 had PV. The detection of an abnormal karyotype in Patients 1 and 2 was an important factor in establishing the diagnosis of acute leukemia. Preliminary evidence supported the suggestion that some chromosomal changes were nonrandom in myeloproliferative diseases. Nonrandom abnormalities involving the same chromosome were observed in several human neoplastic disorders.

3393 CHROMOSOMES AND CAUSATION OF HUMAN CANCER AND LEUKEMIA: X. BANDING PATTERNS IN CANCEROUS EFFUSIONS. (Eng.) Kakati, S. (Roswell Park Memorial Inst., Buffalo, NY 14203); Hayata, I.; Oshimura, M.; Sandberg*, A. A. *Cancer* 36(5): 1729-1738; 1975.

The cells from five cancer effusions (two ovarian carcinomas, two lung cancers, and one carcinoma of the breast) were analyzed (by G-, C-, and Q-banding techniques). The origin of some marker chromosomes could be traced accurately by these banding techniques. Several chromosomes, which appeared normal with conventional staining techniques, were found to be re-arranged and, hence, abnormal. Chromosomes no. 1, no. 3, and no. 11 were the most frequently involved in aberrations, whereas no. 12, no. 13, no. 17-20, and no. 22 were least frequently involved. Only in one case each was the X chromosome or the Y chromosome involved in aberrations. The Y chromosome was found to be missing in all cancer cells of one lung cancer. Each effusion had characteristic markers, invariably present in each cell, whether the cells were near diploid, diploid, or polyploid. No common markers were observed in the two ovarian carcinomas studied (whereas the two lung cancers had a few common markers).

3394 INFLUENCE OF THYROID HORMONES ON SATELLITE ASSOCIATION IN MAN AND THE ORIGIN OF CHROMOSOME ABNORMALITIES. (Eng.) Nilsson, C. (Cancer Chromosome Lab., Inst. Genetics, S-223 62 Lund, Sweden); Hansson*, A.; Milsson, G. *Hereditas* 80(2): 157-166; 1975.

The satellite chromosome association pattern was studied in cells from 6 hyperthyroid patients (before and after antithyroid treatment). The same phenomenon was studied in cells from 4 healthy control individuals without any history of thryoid dysfunction. All analyses were blind studies and were based on 1,200 cells (50 cells in each individual sample). Chromosomes No. 21 and 14 had a significantly higher association tendency when the patients were hyperthyroid. The increase in association tendency of No. 21 and 14 was also found when cells from the control individuals were cultured in medium with serum from hyperthryoid patients. No. 21 and 14 also formed association pairs with each other more frequently when the cells were cultured in medium with excess of the thyroid hormones (thyroxin and triiodothyronin). These hormones could increase the tendency to satellite association of chromosomes 21 and 14, and might therefore also increase the risk of nondisjunction and of translocation between these chromosomes.

3395 CYTOGENETIC EVIDENCE FOR THE SPLENIC ORIGIN OF CHRONIC MYELOID LEUKEMIA. (Fre.) Armenta, D. (Service d'Hematologie, Hotel-Dieu de Montreal, Montreal, Canada); Cadotte, M.; Beaulieu, R.; Neemeh, J.; Long, L.; Pretty, H.; Gosselin, G. *Union Med Can* 105(6): 922-927; 1976.

A case history is presented of a woman with polycythemia vera (PV) of 10 yr duration that evolved into chronic myeloid leukemia (CML). PV was initially diagnosed in 1964 when the patient, age 42 yr, presented with hemoglobin 21.6 g%, hematocrit 71%, splenomegaly, and normal bone marrow. Treatment (bleeding, Leukeran, P^{32}, and vitamin B-12) lowered the hematocrit value to below 50%. A cytogenetic study of the sternal marrow taken in 1971 was normal. Cervical adenopathy appeared in April 1975. The spleen had enlarged to the degree of discomfort. Bone biopsy showed myeloid hyperplasia with megakaryocytosis and slight fibrosis. Malignant lymphoma of the stem cells was disclosed when a cervical lymph node was biopsied; however, the sternal marrow showed no evidence of leukemia. When the spleen was removed, multiple nodules were observed that also proved to be lymphomas of the stem cells. Cytogenetic study of the splenic tissue revealed chromosomal aberrations and the Philadelphia chromosome. The Ph chromosome confirmed a tentative prior diagnosis of CML. At no time was the chromosome seen in the peripheral blood or bone marrow, although it later appeared in a cervical lymph node. The patient was successfully treated for an ensuing blastic crisis. It is concluded that the initial appearance of the Ph chromosome in the spleen supports the theory of a splenic origin for CML. (21 references)

3396 AUTORADIOGRAPHIC AND CYTOPHOTOMETRIC STUDIES ON GRANULOCYTE PRECURSORS IN CHRONIC MYELOID LEUKAEMIA. (Eng.) Quaglino, D. (Instituto di Patologia Medica Policlinico, via del Pozzo, 41100 Modena, Italy); de Pasquale, A.; Zanni, G.; Zagni, G. *Haematologica* 59(3): 298-315; 1974.

Autoradiographic studies with ^{3}H-thymidine and uridine-5-T were carried out on bone-marrow granulocyte precursors from 14 cases of chronic myeloid leukemia and two normal subjects, and their metabolic behavior was compared both in the initial stage and in blastic crisis. Cytostatic treatment had been interrupted at least 2 wk before studies were performed. A combined autoradiographic and cytophotometric study was performed in four cases (two initial stage; two blastic crisis) to correlate the metabolic activities of the granulocyte precursors with their position in relation to the cell cycle as shown by their DNA content. In the cytophotometrical studies, at least 100 cells were analyzed. The autoradiographic data indicated that the labeling index of normal myeloblasts is higher than that of leukemic myeloblasts even though the labeling index in chronic myeloid leukemia cases varies considerably. The labeling index of leukemic myeloblasts was lower than that of promyelocytes in almost all cases, although with different grades of intensity. The relatively higher labeling index of promyelocytes and myelocytes is explained by assuming that, besides the number of nonproliferating and nondifferentiating myeloblasts and a small residual nonleukemic clone, there may exist a granuloblastic leukemic cell line that maintains a good differentiation capacity. Cytophotometric studies indicated that in blastic crisis there is an enhancement of the functional alterations present in small degree in the initial stage of the disease so that the salient feature is a progressive incapacity for differentiation. It is important to establish whether the appearance of cell lines with an altered karyotype parallels the presence of cell lines displaying increasing metabolic alterations. (10 references)

3397 FACTOR-XIII SUBUNITS-A AND -S IN CONGENITAL DEFICIENCY AND IN ACUTE MYELOBLASTIC LEUKEMIA. (Eng.) Barbui, T. (Divisione di Ematologia dell'Ospedale Generale Regionale di Vicenza, Italy); Rodeghiero, F.; Dini, E. *Haematologiga* 59(4): 458-466; 1974.

The two subunits, A and S, of plasma fibrin stabilizing factor (FSF, factor XIII) were assayed in a patient with congenital FSF deficiency, in her relatives, and in several patients with acute myeloblastic leukemia (AML). Factor XIII was assayed in platelet-poor plasma by a method based on the use of antifactor XIII serum in an inhibition test system. Subunits A and S were determined immunologically in agarose gel containing antibodies. Plasma from the patient with the FSF deficiency and her relatives reacted only with anti-S antiserum and not with the anti-A type. In AML patients subunit A was first decreased and then increased to normal when remission had been obtained. No significant changes in subunit S plasma concentration were seen in these patients. Fibrinogen and platelet levels were not correlated with subunit A concentration. There was a positive and statistically significant correlation between subunit A and serum pseudocholinesterase activity, an enzyme considered to be a reliable index of the proteosynthetic function of the liver. Infusion of factor XIII concentrate in a leukemic patient resulted in an immediate rise of subunit A and FSF activity. A gradual reduction of activity and a second rise greater than the initial one occurred after 5 days. After the initial peak, subunit A showed a progressive gradual decline in concentration. It is concluded that homozygote patients are devoid of immunologically identifiable subunit A related to FSF activity. Heterozygote patients, as found in the FSF-deficient patient's relatives, show a decrease in subunit A in accordance with FSF activity. The finding that depressed levels of FSF activity in AML are correlated with subunit A led to the conclusion that congenital and acquired defects of FSF activity are associated with true active protein deficiency. Changes of subunit A might be interpreted as a direct consequence of an impaired synthesis in the liver. (19 references)

3398 THE NON-HODGKIN'S LYMPHOMAS. (Eng.) Dorfman, R. F. In: *The Reticuloendothelial System*. International Acad. of Pathology Monograph. (Baltimore: Williams and Wilkins Co.): pp. 262-281; 1975.

A discussion is presented of the concepts inherent in the classification of the non-Hodgkin's lymphomas by Rappaport in 1966. Recent electron microscopic, immunofluorescent, and membrane receptor studies have cast doubt upon the validity of the concept of a mixed lymphocytic and histiocytic or a predominantly histiocytic type of lymphoma within the spectrum of the nodular lymphomas. They have provided evidence of the origin of nodular lymphomas from follicular B lymphocytes, substantiating the term follicular lymphoma. The need to identify lymphomas with a follicular pattern is stressed because of their favorable prognosis, despite a tendency for early widespread dissemination. An alternative classification is proposed that eliminates controversial terminology, introduces new designations based to some extent on the functional capacity of the component cells, and employs terms appropriate for both diagnostic and therapeutic purposes.

3399 HISTOLOGICAL GRADING, HISTOCHEMISTRY, AND ELECTRON MICROSCOPY OF SCIRRHOUS CARCINOMA OF THE BREAST. (Eng.) Machinami, R. (Faculty Medicine, Univ. Tokyo, Hongo 7-3-1, Bunkyo-ku, Tokyo 113, Japan). *Gann* 67(1): 11-24; 1976.

Twenty-six scirrhous carcinomas of the breast were divided into three histological grades of malignancy: low (grade I), intermediate (grade II), and high (grade III). The correlation of the grades with histochemical and electron microscopic findings in both tumor cells and host tissues was examined. The tumor cells contained increased amounts of lysosomal acid phosphatase and β-glucuronidase. This increase was most marked in grades I and II tumors, and the increase was consistent with lysosomelike fine structures. Both intracytoplasmic lumina and microvilli against stroma were found in the carcinoma cells, and they were mostly found in grades I and II tumors. Segments of intact basal laminae and myoepithelial cells were also found in grades I and II carcinomas. The stroma contained moderately increased amounts of intracellular acid phosphatase and β-glucuronidase, independent of tumor grade. The stroma also contained a large amount of acid mucopolysaccharide ground substance, irrespective of the three grades. There was a striking difference in the ultrastructural organization of the stroma between normal and neoplastic tissues. Although fragmented elastic fibers, increased amount of acid mucopolysaccharide granules, and macrophages rich in phagolysosomes were prominent fine structures of the stroma of carcinomas, there was no apparent difference in them among the three grades of malignancy.

3400 BENIGN CERVICAL AND VAGINAL CHANGES ASSOCIATED WITH EXPOSURE TO STILBESTROL IN UTERO. (Eng.) Sandberg, E. C. (Stanford Univ. Sch. Medicine, Stanford, CA 94305). *Am J Obstet Gynecol* 125(6): 777-789; 1976.

Observations of 320 women who were exposed to stilbestrol in utero showed at least 12 distinct anatomical changes of the cervix and vagina. Five cervical changes, all benign, were found in 57.5% of the women studied. These changes included circular sulcus on the face of the exocervix, recessed areas of perioral tissue, complete covering of the exocervix with endocervical epithelium, pseudopolyp, and anterior cervical protuberances. A greater array of vaginal changes was observed, but they were found to occur in a smaller number of patients (37.8%), were less obvious than cervical changes, and were difficult to discover and interpret. The changes with the greatest frequency of occurrence included: surface adenosis, cystic adenosis, corollalike elevations in the mucosal membrane, fibrous vaginal bands, apical narrowing, and forniceal obliteration. Although other benign changes were observed, they did not warrant classification because of infrequency of appearance or lack of unique identifying characteristics. (17 references)

3401 THE FORMATION OF EMBRYOID BODIES IN VITRO BY HOMOGENEOUS EMBRYONAL CARCINOMA CELL CULTURES DERIVED FROM ISOLATED SINGLE CELLS. (Eng.) Martin, G. R.; Evans, M. J. In: *Teratomas and Differentiation.* Edited by M. I. Sherman and D. Solter. (New York: Academic Press, Inc.): pp. 169-187; 1975.

Embryonal carcinoma cells, the stem cells of teratocarcinomas, are useful in the study of mammalian cell determination and differentiation because they are similar to the pluripotent cells of the early embryo. Embryoid bodies (which resemble the embryonic portion of a 5-day mouse embryo in consisting of an inner core of undifferentiated stem cells surrounded by a single layer of endodermal cells) can be formed in vitro from homogeneous clones of embryonal carcinoma cells by three methods. (a) Plating in the absence of feeder cells permits the gradual formation of rounded clumps in which an outer layer of endodermal cells becomes apparent after detachment from the substratum. (b) Seeding on a nonadhesive substratum results in aggregation in suspension and the similar development of endodermal cell layers around the aggregated cells. (c) Seeding at very low densities (e.g., 100 cells per 90-mm plate) on a feeder layer, removing the small colonies that form, and reseeding as for (b) results in the formation of embryoid bodies for which there is a high statistical probability that each one is itself a clone. The clonal embryonal cell lines were grouped into three distinct categories: (A) nullipotent cells that formed homogeneous aggregates not surrounded by endodermal cells; (B) pluripotent cells that formed embryoid bodies that remained ''simple'', with carcinoma cells and an endodermal layer only; (C) pluripotent cells that initially formed simple embryoid bodies that subsequently progressed to cystic forms in suspension, containing a complex variety of differentiated tissues and a fluid-filled cyst. (29 references)

3402 DIFFERENTIATION OF TERATOMA CELL LINE PCC4.azal IN VITRO. (Eng.) Sherman, M. I. In: *Teratomas and Differentiation.* Edited by M. I. Sherman and D. Solter. (New York: Academic Press, Inc.): pp. 189-205; 1975.

Embryonal teratocarcinoma PCC4.azal cells maintain a uniform morphology under normal tissue culture conditions, although they can form teratomas containing different types of differentiated cells when injected into mice. When grown at high densities after having been plated onto Nucleopore filters or bacterial petri dishes to which they do not attach, the cells form large multicellular balls. Differentiated cells may arise around the periphery of these aggregates after they are replated onto normal plastic tissue culture surfaces, to which most of the cells attach, forming a disk of cells with a dense core. The trigger for cell differentiation appears to be the formation of a three-dimensional matrix of at least 50,000 cells. Once stimulated in this fashion, the undifferentiated PCC4.azal cells within the disk retain the ability to differentiate under normal tissue culture conditions after cloning. A similar three-dimensional matrix of a smaller critical mass may trigger differentiation in the normal embryo. (26 references)

3403 MALIGNANT TRANSFORMATION OF A BENIGN EXOCRINE NEOPLASM OF THE SWEAT GLAND. (Pol.) Kowalewska, Z. (Oddzial Dermatologiczny Szpitala Miejskiego, ul. Malczewskiego 7, 26-600 Radom, Poland). *Przegl Dermatol* 62(6): 797-802; 1975.

A tumor developing from an exocrine sweat gland of the forehead became malignant in an 82-yr-old man after a 10-yr benign course and metastasized to a node in the preauricular region. The disease began in 1964 with a small, slowly expanding tumor on the forehead, which was removed. Numerous mesodermal tubercles covering the forehead and extending to the temple appeared several years later. A small, rapidly growing tumor in the right preauricular region was removed in 1974. Microscopy showed its texture to be similar to that of the primary neoplasm. The patient was discharged in good condition. To date, the existing tubercles have increased only insignificantly. (9 references)

3404 OBSERVATION OF FAMILIAL MEDULLARY CANCER OF THE THYROID. (Fre.) Gautier-Benoit, C. (No affiliation given); Pinchon-Marsy, S.; Calmettes, C.; Houcke, M.; Vermesse, G. *Lille Chir* 30(4): 61-65; 1975.

Three familial medullary cancers of the thyroid (43-yr-old female, her 23-yr-old daughter, and the daughter's 36-yr-old uncle) are presented. The number of familial cases, as opposed to isolated cases, is growing; in a familial case, 50% of the descendants are generally affected. Total thyroidectomy, the recommended treatment, was performed on all three; metastasis and recurrence are suspected in the mother and uncle, respectively. The mother had a pheochromocytoma, which is frequently associated with medullary cancer of the thyroid and which, in these cases, is generally bilateral and multicentric but rarely hypertensive. Elevated calcitonin and histaminase levels are indicative of possible carcinoma or a recurring tumor. Three more members of the 14-person family had elevated calcitonin levels (grandfather, brother, and sister of the daughter). Surgery should be performed on the basis, not of a single elevated calcitonin report, but of continued elevation of calcitonin or detection of a nodule.

3405 PRINCIPAL MULTIPLE CANCERS OF THE COLON. (Eng.) Barna, L. (Semmelweis Medical Univ., Budapest, Hungary); Herczeg, B.; Toth, A. *Am J Proctol* 27(2): 57-64; 1976.

Four case reports of principal multiple colon cancers are presented. The incidence of primary multiple tumors is much higher than previously supposed; statistical data show that in 3%-5% of the pathological cases a double or multiple carcinoma was revealed. A series of 324 patients with colon cancer was seen from 1964-1972; 4 proved to have multiple primary tumors (all adenocarcinomas), an incidence rate of 1.24%. Three patients were women, two over 60 yr and one 32 yr old; the man was 39. A cecal and a rectal adenocarcinoma were present in the man, and in the 32-yr-old woman a tumor developed in the rectum 7 yr after a successful resection. The risk of overlooking a second tumor can be lessened by a very careful irrigoscopy and colonoscopy. Careful abdominal exploration is necessary when a tumor in the lower part of the colon causes such stenosis that the upper portion of the colon cannot be sufficiently filled with contrast medium. Second or subsequent tumors have been successfully surgically treated. The necessity for careful followup of colon cancer patients is, therefore, emphasized. Multiple tumors have a lower survival rate but their course is neither more malignant nor more metastatic than that of single tumors. The risk of overlooking a multiple tumor when colon cancer is diagnosed is stressed. (21 references)

3406 ADENOCARCINOMA OF THE SMALL BOWEL IN ASSOCIATION WITH REGIONAL ENTERITIS: FOUR NEW CASES. (Eng.) Valdes-Dapena, A. (Dept. Pathology, Univ. Pennsylvania, Philadelphia, PA); Rudolph, I.; Hidayat, A.; Roth, J. L. A.; Laucks, R. B. *Cancer* 37(6): 2938-2947; 1976.

Four new cases of adenocarcinoma of the small bowel in association with regional enteritis of long standing are reported. A 48-yr-old man with a 30-yr history suggestive of chronic bowel inflammatory disease developed diffuse infiltrating-type adenocarcinoma of the jejunum. Histologically, there was no clear area of demarcation between enteritis and metaplasia and neoplasia. He died 14 mo after surgery. A 64-yr-old man developed diffusely invasive adenocarcinoma in the terminal ileum 20 yr after a bypass operation for terminal ileitis. Foci of adenocarcinoma intermingled histologically with regions of chronic enteritis. He died 3 mo postoperatively. A 25-yr-old man had been treated intermittently with prednisone (2.5 mg bid over 8 yr) for nonstenosing jejunoileitis. An episode of obvious intestinal obstruction prompted admission 11 yr after the onset of symptoms. At laparotomy, ascites and peritoneal carcinomatosis were observed, which, on histological examination, was adenocarcinoma. The patient died within 10 days. A 47-yr-old man with a 17-yr history of ''ulcerative colitis'' had resection of the terminal small bowel for chronic ileitis. Two years later right lower quadrant pain and tenderness prompted readmission; radiographic examination revealed changes that were described as dilated small intestinal loops and coarse nodularity of the distal ileum. Five years later he developed signs of intestinal obstruction, and at surgery, a stenosing lesion of the mid-jejunum was resected, with a microscopic diagnosis of infiltrating carcinoma. He died 10 mo later with signs of peritoneal carcinomatosis. Although the number of cases of adenocarcinoma with small bowel inflammatory disease is too small for statistical analysis, the association of these conditions has been reported more frequently in recent years. The possible role of immunosuppression from steroid treatment in the etiology of these tumors is discussed. (33 references)

3407 THE CANCER FAMILY SYNDROME AND FAMILIAL CLUSTERING OF SOLITARY COLORECTAL CARCINOMA: SURGICAL AND THEORETICAL CONSIDERATIONS. (Eng.) Smith, W. G. (El Paso Medical Center, 1501 Arizona St. (10-C), El Paso, TX 79902). *Dis Colon Rectum* 19(2): 126-132; 1976.

Cancer Family Syndrome (CFS), a genetically distinct syndrome causally related to neoplasms of the large bowel, has received very little attention in the literature. Several family pedigrees and one case history are presented to discussion of the character of this syndrome. Many genetically related members of a family with CFS develop cancers, frequently at an early age and primarily in the gastrointestinal tract. The environments of all affected members of a CFS family are reviewed and it is concluded that a genetic mechanism is the dominant influence in the development of these malignancies. All genetically related members of a CFS family have a high cancer risk and should have regular and frequent examinations, especially of the large bowel. Once a malignancy has been detected and treated, the assessments should be more frequent and thorough. Study of the CFS should lead to greater understanding of the genetic factors involved in cancer and could be an aid in elucidating the progress of ''normal'' colon cancers.

3408 HISTOLOGICAL CLASSIFICATION OF URINARY BLADDER CANCERS IN RATS INDUCED BY N-BUTYL-N-(4-HYDROXYBUTYL)NITROSAMINE.

(Eng.) Fukushima, S. (Nagoya City Univ. Medical Sch., 1 Kawasumi, Mizuho-cho, Mizuho-ku, Nagoya 467, Japan); Hirose, M.; Tsuda, H.; Shirai, T.; Hirao, K.; Arai, M.; Ito, N. *Gann* 67(1): 81-90; 1976.

Histological types and grades of 613 urinary bladder cancers previously induced in male Wistar rats by N-butyl-N-(4-hydroxybutyl)nitrosamine (BBN) were analyzed. Most of them (95.1%) were transitional cell carcinomas, the remainder being squamous cell carcinomas (3.3%), undifferentiated carcinomas (2.5%), and carcinosarcomas (0.3%). Among the transitional cell carcinomas, 23.3% were Grade I anaplasia, 55.2% Grade II, and 21.5% Grade III. Among the squamous cell carcinomas, 20% each were Grade I and II, and 60% were Grade III. Most of the undifferentiated carcinomas were Grade III. Some of the cases of transitional cell carcinoma had areas of squamous metaplasia and/or glandular metaplasia, and the incidence of metaplasia increased with the grade of anaplasia. Approximately 36% of the cases of transitional cell carcinoma were of the invasive type. Invasive types were twice as numerous among cases of squamous cell carcinoma as among those of transitional cell carcinoma, and all of undifferentiated carcinomas were invasive. The incidence of invasive type was closely related to the grade of anaplasia. These results show that the morphological characteristics of urinary bladder carcinomas induced by BBN in rats are similar to those seen clinically in humans.

3409 RENAL ANGIOMYOLIPOMA IN A CASE OF TUBEROUS SCLEROSIS, AN ELECTRON MICROSCOPY STUDY.

(Eng.) Sun, C. N. (Veterans Admin. Hosp., Little Rock, AR 72206); White, H. J.; Bissada, N. K. *Beitr Pathol* 156(4): 401-410; 1975.

A case history, including electron microscopic findings, of renal angiomyolipoma with tuberous sclerosis is presented and possible origins of the tumor cells are suggested. Two wk prior to admission, the patient (a 23-yr-old male) had an acute onset of right colicky flank pain and gross hematuria. Pertinent physical findings were limited to the skin. Adenoma sebaceum was present along the nasolabial furrow and extended into the medial portion of the cheeks; several fibrous nodules were on the forehead, hypopigmented maculae were found on the chest and back, and multiple periungual fibromas on the feet. Renal arteriography demonstrated bilateral vascular renal tumors which were diagnosed as renal angiomyolipoma by open biopsy. The postoperative course was uneventful. Electron microscopic examination of the biopsy specimens revealed two types of cells, smooth muscle cells and what appeared to be fibroblastic or parenchymal cells. The smooth muscle cells were loosely arranged, contained myofilaments, large indented nuclei, rough endoplasmic reticulum, mitochondria, dense granules, fat vacuoles, and an unusual amount of glycogen granules. In many of the smooth muscle cells showing degenerated myofilaments and microtubules, there appeared to be an increased amount of glycogen granules. Golgi fields were identified. In the fibroblastic or parenchymal cells, the nuclei were frequently lobed and showed nuclear or cytoplasmic inclusions. The endoplasmic reticulum occasionally had a whorllike arrangement. There were abundant small lysosomal granules and a large amount of glycogen granules. Golgi fields were also present. Although a few fibroblastic cells were found, the majority of tumor cells were either smooth muscle type or a degenerated form thereof.

3410 ALPHA 1-ANTITRYPSIN PHENOTYPES IN CIRRHOSIS AND HEPATOMA.

(Eng.) Theodoropoulos, G. (Sch. Medicine, Univ. Athens, Greece); Fertakis, A.; Archimandritis, A.; Kapordelis, C.; Angelopoulos, B. *Acta Hepatogastroenterol (Stuttg)* 23(2): 114-117; 1976.

To ascertain the prevalence of α1-antitrypsin deficiency in cirrhosis patients and to verify whether homozygous or heterozygous deficiency predisposes one to develop cirrhosis or hepatoma, the phenotypic distribution of α1-antitrypsin variants was studied in 101 patients with cirrhosis and in 50 with cirrhosis plus hepatoma; 504 healthy Greeks served as controls. The ZZ and MZ phenotypes were found only once in the cirrhosis group. The very low PiZ gene frequency suggests that the association of PiZ gene with cirrhosis is fortuitous. The FM phenotype was observed in 14% of patients with hepatoma derived from cirrhosis; this incidence differed significantly between the two groups of patients and the controls. It is possible that a high percentage of phenotypically FM cirrhosis patients develops hepatoma; the reason is unknown.

3411 MORPHOLOGY OF SPONTANEOUS AND INDUCED NEOPLASIA.

(Eng.) Jones, G.; Butler, W. H. In: *Mouse Hepatic Neoplasia. Proceedings of a Workshop held at the H.T.S. Management Centre, Lane End, High Wycombe (Great Britain), 12-17 May 1974.* (New York: Elsevier Scientific Publishing Co.): pp. 21-59; 1975.

The morphology of the four main groups of spontaneous and induced liver lesions (parenchymal, vascular, tumors arising from the bile duct, and others, such as lymphoreticular lesions) in mice is described. The lesions were induced by treating the mice with phenobarbitone, dimethylnitrosamine, DDT, dieldrin, scarlet red, and acetylaminofluorene. Parenchymal cell nodules vary from 1-2 mm to several centimeters in diameter; multiple lesions are found in a single liver and occur in all lobes. These lesions have been classified into two histological types: type A, a simple nodular growth of parenchymal cells that are difficult to distinguish from the adjacent normal liver; and type B, nodules of parenchymal cells showing areas of papilliform and adenoid growth. The cells from induced type A or B nodules are larger than those from untreated mice, and many stain intensely eosinophilic. The nuclei vary in size and many are 4-5 times normal size with multiple nucleoli, inclusions, and infoldings. Both spontaneous and compound-induced lesions metastasize, but the incidence in all series is very low. Vascular nodules arising in the mouse liver cover a wide range of morphological types. In some cases, the liver may be enlarged 2-3 times normal size by multiple blood-filled cysts surrounded by normal or compressed liver parenchyma. Cellular proliferation, granulation tissue at the edge of the cysts and associated with thrombus or clot, and transplantability have led to this lesion being considered neoplastic. A highly malignant vascular lesion seen in multiple sites (liver, lung, kidney, and spleen) is best described as an angiosarcoma. Cholangiomas consist of sheets of uniform cells with large dark nuclei, at first lining cystic spaces and later forming solid tumors. The possibility that these lesions are mesenchymal in origin has been suggested. The most common secondary tumor found in mouse liver is the reticuloendothelial tumor in which the proliferating cells form solid masses around the portal tracts and central veins. There does not appear to be a good correlation between the morphological appearance of the lesion and its biological behavior in treated mice. (24 references)

3412 ASSOCIATION OF PHEOCHROMOCYTOMA AND NEUROFIBROMATOSIS: THREE PERSONAL OBSERVATIONS. (Fre.) Veyre, B. (Hopital Cardio-vasculaire et Pneumologique, 108, bd Pinel, F 69394 Lyon Cedex 3, France); Saint-Pierre, A.; Laffet, G.; Milon, H.; Sassolas, G.; Perrin, A. *Nouv Presse Med* 4(40): 2873-2876; 1975.

Three cases of associated neurofibromatosis and pheochromocytoma (one woman and two men, 27, 55, and 33 yr, respectively) are presented. The frequency, sex, and age of appearance of each disease is given singly and in association; familial forms, clinical manifestations, sites of the pheochromocytomas, diagnostic stages, associated conditions, Sipple syndrome, and pathogenesis are reviewed.

3413 AN ULTRASTRUCTURAL STUDY OF NUCLEAR AND CENTRIOLAR CONFIGURATIONS IN MULTINUCLEATED GIANT CELLS. (Eng.) Sapp, J. P. (Div. Oral Pathology, Univ. Western Ontario, Ontario, London, Canada). *Lab Invest* 34(2): 109-114; 1976.

To investigate the location and arrangement of the nuclei and centrioles in the multinucleated cells of a variety of lesions and tissue found in both humans and experimental animals, multinucleated giant cells were examined with electron microscopy from (1) four peripheral giant cell granulomas of the jaws, (2) a central giant cell tumor of the maxilla, (3) five giant cell tumors of tendon sheath, (4) experimentally induced foreign body granulomas in rats, (5) a virus-induced sarcoma, and (6) osteoclasts from the mandibles of rats and (7) the femurs of hamsters. This study was conducted: (1) to examine the location and arrangement of the nuclei and the centrioles of the multinucleated cells and compare them with the osteoclast which was formerly thought to have a unique arrangement and (2) to observe whether the physical location of the centrioles and the nuclei were such that the nuclei could undergo mitosis within the cytoplasm. All of the multinucleated cells were found to have a similar arrangement of nuclear concentration areas and nucleus-free areas. A giant centrosphere containing multiple pairs of centrioles was found in the nucleus-free areas, unassociated with any particular nucleus. In the case of the foreign body giant cell and the osteoclast, this giant centrosphere was located very close to the foreign material or bone. When single pairs of centrioles were found, they were located in the area of nuclear concentration, closely associated with a particular nucleus at the periphery. These findings have shown that a common centrosphere containing multiple centrioles is not an exclusive feature of the osteoclast as was previously thought. The findings suggest that a mononuclear cell containing a centriole pair fuses to the larger cell and maintains its centriole pair in close proximity to its nucleus for a short period of time, during which it may undergo mitotic activity. Eventually, the centrioles proceed to a common centrosphere whereas the nuclei aggregate in another area making further mitotic activity an unlikely possibility.

3414 MUTATIONS. PART I: GENETIC MUTATIONS AND HEREDITARY DEFECT. (Fre.) Auffret van der Kemp, Th. (No affiliation given). *Revue du Palais de la Decouverte* 4(34): 43-56; 1976.

3415 GENETIC CONTROL OF CELL DIFFERENTIATION IN PLATYFISH-SWORDTAIL MELANOMAS. (Eng.) Vielkind, U. (Genetisches Institut der Justus Liebig-Universitaet, D-63 Giessen, W. Germany). *J Exp Zool* 196(2): 197-204; 1976.

3416 THE THERAPEUTIC IMPLICATIONS OF SPLENIC INVOLVEMENT IN STAGE IIIA HODGKIN'S DISEASE (HD) (Proceedings Abstract). (Eng.) Levi, J. A. (NCI-BCRC, Baltimore, MD 21201); Wiernik, P. H. *Proc Am Assoc Cancer Res* 17: 56; 1976.

3417 ULTRASTRUCTURE OF HUMAN BREAST HISTOCULTURES (Proceedings Abstract). (Eng.) Richters, A. (Univ. Southern California, Los Angeles, CA 90033); Sherwin, R. P. *Fed Proc* 35(3): 410; 1976.

3418 FREE AMINO ACIDS IN CANCER TISSUE (Proceedings Abstract). (Eng.) Hernandez, T. (Louisiana State Univ. Medical Center, New Orleans, LA 70112); Coulson, R. A.; Morgan, L. R.; Herbert, J. D. *Fed Proc* 35(3): 410; 1976.

3419 THE MORPHOLOGY OF TERATOMATOUS ORGANS IN TISSUE CULTURES OF *TARAXACUM OFFICINALE* WEBER. (Eng.) Bowes, B. G. (Glasgow, Scotland). *Cellule* 71(1): 21-29; 1975.

3420 OBSTRUCTIVE JAUNDICE IN A PATIENT WITH EPIDERMOID CARCINOMA OF THE HYPOPHARYNX. (Eng.) Senior, R, (Depts. Internal Medicine and Pathology, Washington Univ. Sch. Medicine, St. Louis, MO); Melson, G. L.; Session, D.; Lynch, R. *Am J Med* 60(5): 687-695; 1976.

3421 LEWANDOWSKY-LUTZ SYNDROME (Abstract). (Fre.) Kaufmann, J. (Zurich, Switzerland); Meves, C.; Storck, H. *Bull Soc Fr Dermatol Syphiligr* 82(3): 272-273; 1975.

3422 EPITHELIO-RETICULAR CELL THYMOMA WITH LYMPHOCYTIC ''EMPERIPOLESIS'': AN ULTRASTRUCTURAL STUDY. (Eng.) Llombart-Bosch, A. (Dept. de Histologia y Anatomia Patologica, Facultad de Medicina, Paseo al Mar, Valencia-10, Spain). *Cancer* 36(5): 1794-1803; 1975.

3423 PLEOMORPHIC ADENOMA OF THE SALIVARY GLAND: WITH SPECIAL REFERENCE TO HISTOCHEMICAL AND ELECTRON MICROSCOPIC STUDIES AND BIOCHEMICAL ANALYSIS OF GLYCOSAMINOGLYCANS IN VIVO AND IN VITRO. (Eng.) Takeuchi, J. (Sch. Dentistry, Aichi-Gakuin Univ., 2-11, Suemori-dori, Chikusa, Nagoya, Japan); Sobue, M.; Yoshida, M.; Esaki, T.; Katoh, Y. *Cancer* 36(5): 1771-1789; 1975.

3424 ON THE ULTRASTRUCTURE OF HIBERNOMA. (Eng.) Seemayer, T. A. (Montreal Children's Hosp., 2300 Tupper St., Montreal, H3H 1P3 Canada); Knaack, J.; Wang, N.-S.; Ahmed, M. N. *Cancer* 36(5): 1785-1793; 1975.

3425 SQUAMOUS METAPLASIA IN THE HEALING OF CHRONIC COLONIC ULCERS OF THE RAT. (Eng.) Reeve, D. R. E. (Royal Coll. Surgeons England, Lincoln's Inn Fields, London WC2A 3PN, England). *J Pathol* 117(1): 15-22; 1975.

3426 FECAL BACTERIOLOGY OF COLONIC POLYP PATIENTS AND CONTROL PATIENTS. (Eng.) Finegold, S. M. (Univ. California Los Angeles Sch. Medicine, Los Angeles, CA 90024); Flora, D. J.; Attebery, H. R.; Sutter, V. L. *Cancer Res* 35(11/Part 2): 3407-3417; 1975.

3427 PERIFOLLICULAR FIBROMATOSIS CUTIS WITH POLYPS OF THE COLON--A CUTANEO-INTESTINAL SYNDROME SUI GENERIS. (Eng.) Hornstein, O. P. (Dermat. Univ.-Klinik, D-8520 Erlangen, W. Germany); Knickenberg, M. *Arch Dermatol Res* 253: 161-175; 1975.

See also:

* (Rev): 3001, 3003, 3026, 3034, 3035, 3036, 3037, 3038, 3039, 3040, 3041, 3042, 3043, 3044, 3045, 3056, 3057
* (Chem): 3071, 3073, 3074, 3098, 3100, 3102
* (Phys): 3144
* (Viral): 3167, 3174, 3181, 3184, 3185, 3206, 3241, 3259, 3275, 3284, 3290, 3311, 3314, 3322, 3324
* (Immun): 3332, 3335, 3380, 3386, 3388
* (Epid-Biom): 3428, 3433

3428 MALIGNANT MELANOMA OF THE HAND AND FOOT. (Pol.) Wojnerowicz, C. (Wojewodski Szpital Onkologiczny, ul. Garbary nr 15, 61-866 Poznan, Poland); Brzezinski, J.; Milanowski, L. *Przegl Dermatol* 63(1): 1-9; 1976.

Eighty-one malignant skin melanomas of the feet (66) and hands (15) were observed among 479 cases of skin melanomas. Malignant melanomas of the feet and hands were more frequent in women than in men (2:1); the average age of the patients was 49 yr. Neoplasms were secondary to a nevus in 64% of cases of foot melanoma and in 45% of cases of hand melanoma. Surgery was indicated in 42/66 foot melanomas (up to 32/42 with histologically confirmed metastases to lymph nodes) and in 9/15 hand melanomas. In foot melanomas, 5-yr survivals were obtained in 6/8 cases with unconfirmed metastases to the lymph nodes and 2/18 cases with metastases to the inguinal lymph nodes. Five-year survival was obtained in 3/7 hand melanomas, all of them without metastases to the lymph nodes. (19 references)

3429 INCIDENCE OF CHILDHOOD CANCER IN FINLAND. (Eng.) Teppo, L. (Finnish Cancer Registry, Liisankatu 21 B, 00170 Helsinki 17, Finland); Salonen, T.; Hakulinen, T. *J Natl Cancer Inst* 55(5): 1065-1067; 1975.

Between 1953 and 1970, 2,605 malignant tumors in children under 15 yr of age were reported to the Finnish Cancer Registry, a population-based registry covering the whole country. The mean annual age-adjusted incidence rates per million were 128 in males and 108 in females. The most common neoplasms were leukemia (age-adjusted incidence rates, 43.7 in males; 34.7 in females), brain tumors (26.4 in males, 22.8 in females), renal tumors (10.0 in males, 9.1 in females), lymphomas (10.8 in males, 5.3 in females), and bone tumors (5.3 in males, 5.1 in females). This distribution was approximately the same as that observed in many other white populations. The incidence rates of leukemia, lymphomas, neuroblastomas, and soft-tissue tumors were somewhat lower than the figures reported in the Third National Cancer Survey of the United States.

3430 TUMORS IN STILLBORN INFANTS AND IN CHILDREN DURING THE FIRST YEAR OF THEIR LIVES IN CZECHOSLOVAKIA. (Cze.) Kucera, J. (Ustav pro peci o matku a dite, Prague, Czechoslovakia); Peleskova, A. *Cesk Pediatr* 30(9): 421-424; 1975.

Among a total of 2,423,723 consecutive births in the Czech Socialist Republic (1959-1971) and the Slovak Socialist Republic (1961-1969), 456 tumors were diagnosed in stillborn infants and in infants during the first year of life. This was 18-20 cases per 100,000, although the incidence in the Slovak Socialist Republic was somewhat higher, primarily due to leukemia and tumors of the central nervous system (CNS) and adrenals. After the total was reduced by the 69 cases of surviving children with peripheral vessel and skin tumors, the tumor incidence was 15-17 per 100,000 births. The incidence of leukemia and peripheral vessel tumors was seven times higher, and that of CNS and skin tumors was four times higher, in children of mothers aged 35-39 compared with those of mothers aged up to 19. The presence of tumors or birth defects in relatives was greatest for children with CNS tumors and leukemia. The gestation period was approximately normal for children with leukemia and with CNS, respiratory system, and urinary system tumors, while it was 36 wk or less in over 30% of the children with skin and digestive system tumors. In children with leukemia and tumors of the urinary system, adrenals, peripheral vessels, and sense organs, birth weight was normal, but in those with digestive system, oral cavity, and facial tumors, it was severely subnormal. Incidence of brain, mediastinal, and heart tumors was higher in boys, while abdominal, bone, facial, oral cavity, and skin tumors were more frequent in girls. Birth defects most often accompanied digestive system, respiratory system, and skin tumors. Tumor frequency was inversely related to altitude, being lowest at 1,500 m above sea level. It seems that leukemia and tumors of the skin, CNS, gastrointestinal tract, and peripheral vessels are determined prezygotically, while heart and uropoietic system tumors are primarily the result of external factors.

3431 AIR POLLUTION AND CANCER: AN ECONOMETRIC APPROACH. (Eng.) Koshal, R. K. (Ohio Univ., Athens, OH); Koshal, M. *Clean Air* 10(2): 19-22; 1976.

The quantitative relation between cancer mortality rates and air pollution levels and other socioeconomic variables was investigated with the use of data from 39 cities for the years 1960-1967. Mortality rates were assumed to be a function of level of suspended particulate matter, level of benzene-soluble organic matter, population density, humidity, and the percentage of people 65 yr old and over. The analysis suggested that both past and present levels of pollution are associated with cancer mortality, and a 100% increase in air pollution would be accompanied by a 24%-27% increase in cancer mortality rates. Similar findings have been reported for other diseases. An average improvement of about 50% in the air pollution would imply a social savings of the order of 787-886 million dollars per year in terms of cancer alone. (19 references)

3432 MEDICAL SURVEILLANCE OF VINYL CHLORIDE WORKERS. (Eng.) Duck, B. W. (Occupational Health Unit, BP Research Centre, Sunbury on Thames, TW167LN, England). *Proc R Soc Med* 4(69): 307-309; 1976.

The progress of teams set up in England to study workers exposed to vinyl chloride and polyvinyl chloride is reviewed. Although all the results are not yet available, the low incidence of liver disorders in these workers does not seem to warrant the mass screening that was initially undertaken. With stringent environmental standards now in force, future efforts will concentrate on the evaluation of specific hypotheses.

3433 RISK FACTORS ENCOUNTERED IN CASES OF CERVICAL CANCER AND IN PRECARCINOMATOUS CONDITIONS.

(Cze.) Kralova, A. (Gynek.-porod. klinika ILF Praha, Prague, Czechoslovakia). *Cesk Gynekol* 40(7): 551-559; 1975.

The occurrence of risk factors responsible for the development of cervical cancer was studied in 150 women (average age 46.2 yr) with uterine cervical carcinoma, 100 women (average age 37 yr) with conditions precancerous in the strict sense, and in 150 female controls. Educational level was distinctly higher in the precancerous group than in the others. The age of onset of regular sexual activity was 18-19 yr in the majority of the carcinoma group, 20 yr in most of the precancerous group, and 20-21 yr in the majority of controls, while the age of the first pregnancy was most often 18-19 yr in the carcinoma group, 21 and 23 yr in the precancerous group, and 23 yr in the controls. Differences in the number of births were found between the carcinoma group and the other two groups, and the precancerous group had more sexual partners. No significant differences in nervous and emotional instability were found among the studied groups, but the carcinoma group predominated in the category of instability associated with psychological trauma. One-third of the precancerous group were heavy smokers, compared with one-fourth of the carcinoma group. Although the above-mentioned differences were found at varying levels of statistical significance, no reliable predisposing factors were proved in the majority of those in the precancerous and carcinoma groups. Thus, no definite risk groups for cervical cancer could be established.

3434 CANCER MORTALITY IN THE STEEL INDUSTRY.

(Eng.) Radford, E. P. (Johns Hopkins Univ. Sch. Hygiene and Public Health, Baltimore, MD 21205). *Ann NY Acad Sci* 271: 228-238; 1976.

The cancer mortality experience in a steel mill and a shipyard for 1973-1974 was analyzed. Among white shipyard workers, cancer deaths were significantly elevated, with half due to lung cancer; asbestos exposure is the likely causative factor. White steel mill workers had cancer deaths well above the expected (one-third from lung cancer), whereas black workers had a nonsignificant elevation. No bladder cancers were observed in blacks, but significantly elevated kidney and liver and biliary cancers were found. Employees involved in finishing operations had a higher overall cancer mortality than those working on basic steel operations. The lung and ''all other'' cancer categories comprised the excess mortality in steelworkers. A comparison with geographic estimates made by the National Cancer Institute revealed a slight, nonsignificant excess in kidney cancer but a highly elevated bladder cancer ratio; 6 of the 10 bladder cancer cases worked in basic steel operations. The remaining cancers comprising the excess mortality included malignant melanoma, retroperitoneal sarcoma, brain tumor, spinal cord tumor, tumor around the trachea, peritoneal cancer, carcinomatosis, and metastatic cancer. No obvious potential carcinogens could be found in the steelworks.

3435 HOUSEHOLD-CONTACT ASBESTOS NEOPLASTIC RISK.

(Eng.) Anderson, H. A. (Mount Sinai Sch. Medicine, The City Univ. New York, New York, NY 10029); Lilis, R.; Daum, S. M.; Fischbein, A. S.; Selikoff, I. J. *Ann NY Acad Sci* 271; 311-323; 1976.

An attempt was made to assess the risks of nonmalignant and malignant disease associated with household exposure to occupationally derived asbestos dust by investigating 1,664 workers employed at a factory where well-documented asbestos disease was reported from 1941 to 1954. All household contacts living with them at that time are being traced and examined; this report examines the preliminary data. Among the contacts traced, two cases of death due to pleural mesothelioma were found. In addition, two living cohort members have biopsy-proved mesotheliomas and are under therapy. A clinical study was concurrently run with the mortality investigations. Of 326 otherwise healthy household contacts, 35% had chest x-ray abnormalities (pleural and/or parenchymal) characteristic of asbestos exposure. Pleural fibrosis and calcification without parenchymal fibrosis were seen more frequently than parenchymal fibrosis alone. It was concluded that household contamination with asbestos from industrial sources commonly occurs, and it may result in radiological changes and, less frequently, asbestos associated neoplastic disease.

3436 ROLE OF LIFE-STYLE AND DIETARY HABITS IN RISK OF CANCER AMONG SEVENTH-DAY ADVENTISTS.

(Eng.) Phillips, R. L. (Loma Linda Univ. Sch. Health, Loma Linda, CA 92354). *Cancer Res* 35(11/Part 2): 3513-3522; 1975.

Comparison of the mortality experience of Seventh-Day Adventist and non-Seventh-Day Adventist physicians showed equal cancer mortality, consistent with the hypothesis that the apparent reduced risk of cancer death in all Adventists might be due to selective factors. The results of a small case-control study of colon and breast cancer among Adventists showed significant relative risks for colon cancer of 2.8 for past use of meat. For current food use, the significant relative risks were 2.3 for beef, 2.7 for lamb, and 2.1 for a combined group of highly saturated fat foods. The lacto-ovo-vegetarian diet might protect against colon cancer. However, the evidence linking diet to breast cancer was less clear. Due to the marked variability in dietary habits within the Seventh-Day Adventist population, they were thought to be a productive group for further study of cancer and diet.

3437 NEOPLASIA IN THE WOOD AND PULP INDUSTRY.

(Eng.) Milham, S., Jr. (Washington State Dept. Social and Health Services, Olympia, WA 98504). *Ann NY Acad Sci.* 271: 294-300; 1976.

The occupational mortality of white men in the wood products industry in Washington State from 1950 through 1971 was analyzed. A significant elevation in the proportionate mortality ratio (PMR) for stomach cancer was seen in loggers, plywood workers, and in carpenters. Stomach cancer showed a modest PMR increase in all groups, suggesting that ingested or inhaled and swallowed wood particles may cause gastric cancer. The PMR's for small intestine cancer and for multiple myeloma were significantly elevated in pulp and paper makers only. Hodgkin's disease accounted for an excess PMR in pulp and paper makers and in carpenters. A significant increase in the leukemia PMR was seen in plywood workers. The PMR for all malignant neoplasms was significantly elevated in loggers and carpenters. The observed increases in the hematopoietic cancer PMR may indicate that the physical and chemical breakdown products of wood may be carcinogenic. The major negative finding was that men who work in sawmills where wood is simply machined do not show a striking neoplasia excess, as seen in the paper, pulp, and plywood industries.

3438 SURVEILLANCE FOR CARCINOGENESIS FROM STEROID CONTRACEPTIVES. (Eng.) Paffenbarger, R. S., Jr. In: *Host Environment Interactions in the Etiology of Cancer in Man. Proceedings of a Meeting Held at Primosten, Yugoslavia, 27 August-2 September 1972.* International Agency for Res. on Cancer and John E. Fogarty International Center of the Natl. Insts. of Health USA. (Lyon, France): pp. 183-195; 1973.

A discussion of surveillance for carcinogenesis from steroid contraceptives includes a literature review, a discussion of problems associated with various surveillance programs, and a presentation of cancer mortality and incidence data. Figures illustrating the trends of age-specific death rates for cancer of the female breast, ovary, and uterus in California between 1950 and 1970 show no significant increase that would imply an adverse effect from hormone usage. The age-adjusted incidence rates for cancer by race in one county in California (1960-1969) showed that rates for breast, ovary, and uterine cancers were highest for whites (among Negroes, Japanese-, and Chinese-Americans); the rates for cervical cancer were highest for Negroes. Incidence rates for breast cancer in white women of those age groups in which steroid contraceptives may have been used (since their introduction in the late 1950's) demonstrated no trend in age-adjusted rates. Practical problems encountered in case-control and prospective studies are discussed.

3439 EXPERIMENTAL EVIDENCE OF DIETARY FACTORS AND HORMONE-DEPENDENT CANCERS. (Eng.) Carroll, K. K. (Dept. Biochemistry, Univ. Western Ontario, London, Ontario, Canada N6A 5C1). *Cancer Res* 35(11/Part 2): 3374-3383; 1975.

The current awareness of the importance of environmental factors (such as diet) in the etiology of human cancer stimulated renewed interest in animal models for studying effects of diet on tumorigenesis. Diet could influence cancer in animals by affecting the initiation or subsequent preneoplastic stage of tumorigenesis, but it had less effect on tumor growth. Caloric restriction had a general inhibitory influence on tumorigenesis. Dietary fat, on the other hand, tended to promote tumorigenesis; however, only certain types of tumors (such as mammary tumors) were affected. Both caloric restriction and dietary fat appeared to act primarily during the preneoplastic stage; their effects on hormone-dependent tumors might be mediated through changes in the hormonal environment. Variations in other dietary factors (such as protein, vitamins, or minerals) above the levels required for normal maintenance seemed to have little influence on the genesis or growth of tumors.

3440 DIETARY FACTORS AND ESOPHAGEAL CANCER IN THE CASPIAN LITTORAL OF IRAN. (Eng.) Hormozdiari, H. (Univ. Nebraska Medical Center, Omaha, NB 68105); Day, N. E.; Aramesh, B.; Mahboubi*, E. *Cancer Res* 35(11/Part 2): 3493-3498; 1975.

An attempt was made to relate the distribution of exogenous factors to the varying incidences of esophageal cancer in the Caspian Littoral of Iran. A total of 38 villages was chosen by random sampling in 14 regions defined by their esophageal cancer incidence and environmental characteristics. Information was obtained on the dietary, social, and cultural features of each village. Furthermore, an extensive 5-day study of 6 randomly selected households in each of the 38 villages was conducted. The study consisted of measured dietary intake, a historical food consumption questionnaire, and clinical examinations of adult occupants. Results showed no single factor responsible for the etiology of esophageal cancer. There were, however, some major dietary differences between the regions of different esophageal cancer risk. Bread was the chief staple food in high-incidence areas; rice, in low-incidence areas. In high-incidence areas, there was a low intake of vitamins A and C, riboflavin, animal protein, and fresh vegetables and fruit, but a greater consumption of sheep's and goat's milk. Analyses of food samples for aflatoxins, polycyclic aromatic hydrocarbons, and nitrosamines showed low levels of these carcinogens (in areas of high and low incidences). The use of tobacco and alcohol was of no significance.

3441 THE ASSOCIATION OF ECONOMIC STATUS WITH THE OCCURRENCE OF LUNG CANCER. (Eng.) Brown, S. M. (2120 Berkeley Way, Berkeley, CA 94704); Selvin, S.; Winkelstein, W., Jr. *Cancer* 36(5): 1903-1911; 1975.

Lung cancer in men was shown to be inversely associated with economic class (by analysis of 1959-61 mortality rates from Buffalo, New York). Using five economic strata, white men below and above age 55 in the poorest group had rates 2.6 and 3.8 times higher than those in the richest. The trend was not significant for women. The question investigated was whether, for men, an economic-class trend for smoking existed paralleling that for lung cancer. Individuals from a Buffalo sample survey were classified according to cigarette smoking habits. Age was found highly associated with both smoking and economic class. In data from white men stratified for age, no significant association was observed between smoking and economic class. In women, the upper economic groups smoked more (particularly those above age 55). Therefore, a strong inverse economic class gradient for lung cancer in men could not be explained by class-related cigarette smoking patterns alone.

3442 RACIAL AND OCCUPATIONAL VARIATIONS IN CANCER OF THE TESTIS: SAN FRANCISCO, 1956-65. (Eng.) Mustacchi, P. (Dept. International Health, 1699 HSW, Univ. California, San Francisco, CA 94143); Millmore, D. *J Natl Cancer Inst* 56(4): 717-720; 1976.

Correlations among the broad racial and socioeconomic variables in the distribution of cancer of the testis were studied in relation to the 91 residents of San Francisco who were diagnosed with the disease between 1956 and 1965. Of the cases, only two occurred in blacks and two in Orientals; the rest were found in whites. The lower risk observed for nonwhites could not be attributed entirely to genetic factors, since among whites a risk gradient seemed to correlate directly with broad socioeconomic indices such as occupation and census tract of residence. These findings are compatible with a hypothesis that certain occupational groups may have become epidemiologic indicators of testicular carcinogens in a changing environment.

3443 EPSTEIN-BARR VIRUS BEHAVIOR IN DIFFERENT POPULATIONS AND IMPLICATIONS FOR CONTROL OF EPSTEIN-BARR VIRUS-ASSOCIATED TUMORS. (Eng.) de-The, G. (Unit Biological Carcinogenesis, International Agency for Res. on Cancer, 150 Cours Albert Thomas, 69008 Lyon, France). *Cancer Res* 36(2): 692-695; 1976.

The epidemiology of Epstein-Barr virus (EBV) infection in populations at different risk for EBV-associated diseases indicate significant differences between the populations. EBV infection takes place much earlier in Uganda, where all children are infected before the age of 2 to 3 years, than in Southeast Asia, where nasopharyngeal carcinoma is prevalent. The epidemiology of BL suggested the intervention of a strong environmental factor relatively close in time to the clinical expression of the disease, whereas that of NPC stressed the importance of genetic factors. It is proposed that such early infection in Equatorial Africa is related to the risk for Burkitt's lymphoma. Four interventions to control EBV-associated diseases are possible: (a) simple hygienic measures to delay natural primary infection by EBV; (b) EBV vaccine; (c) intervention against cofactors such as malaria in Burkitt's lymphoma; and (d) characterization of high-risk groups to allow early detection and successful treatment.

3444 GEOGRAPHIC PATTERNS OF CANCER MORTALITY IN THE UNITED STATES. (Eng.) Hoover, R.; Mason, T. J.; McKay, F. W.; Fraumeni, J. F., Jr. In: *Persons at High Risk of Cancer. An Approach to Cancer Etiology and Control.* Fraumeni, J. F., Jr., ed. (New York: Academic Press, Inc.): pp. 343-359; 1975.

3445 DEMOGRAPHIC PATTERNS OF CANCER INCIDENCE IN THE UNITED STATES. (Eng.) Cutler, S. J.; Young, J. L., Jr. In: *Persons at High Risk of Cancer. An Approach to Cancer Etiology and Control.* Fraumeni, J. F., Jr., ed. (New York, Academic Press, Inc.): pp. 307-342; 1975.

3446 LUNG CANCER AT AUTOPSY IN A-BOMB SURVIVORS AND CONTROLS, HIROSHIMA AND NAGASAKI, 1961-1970: II. SMOKING, OCCUPATION, AND A-BOMB EXPOSURE. (Eng.) Ishimaru, T. (Editorial Office, Radiation Effects Res. Foundation, Atomic Bomb Casualty Commission, 5-2 Hijiyama Park, Hiroshima, Japan 730); Cihak, R. W.; Land, C. E.; Steer, A.; Yamada, A. *Cancer* 36(5): 1723-1728; 1975.

3447 INTERDISCIPLINARY AND EXPERIMENTAL APPROACHES: METABOLIC EPIDEMIOLOGY. (Eng.) Wynder, E. L.; Hoffman, D.; Chan, P.; Reddy, B. In: *Persons at High Risk of Cancer. An Approach to Cancer Etiology and Control.* Fraumeni, J. F., Jr., ed. (New York: Academic Press, Inc.): pp. 485-501; 1975.

3448 DIETARY PATTERNS AMONG MEN OF JAPANESE ANCESTRY IN HAWAII. (Eng.) Hankin, J. H. (Natl. Cancer Inst. and Sch. Public Health, Univ. Hawaii, Honolulu, HI 96822); Nomura, A.; Rhoads, G. G. *Cancer Res* 35(11/Part 2): 3259-3264; 1975.

3449 FLUORIDATION OF PUBLIC WATER SYSTEMS AND CANCER DEATH RATES (CDRS) IN HUMANS (Proceedings Abstract). (Eng.) Yiamouyiannis, J. A. (Natl. Health Federation, Monrovia, CA 91016); Burk, D. *Fed Proc* 35(7): 1707; 1976.

3450 NUCLEAR MAGNETIC RESONANCE PATTERNS OF CELLULAR WATER IN SYNCHRONIZED HELA CELLS (Proceedings Abstract). (Eng.) Beall, P. T. (Dept. Physiology, Baylor Coll. Medicine, Houston, TX 77025); Rao, P. N.; Huggins, R. A.; Hazlewood, C. F. *Fed Proc* 35(3): 410; 1976.

3451 FACILITATED DIFFUSION OF THYMIDINE INTO MAMMALIAN CELLS IN SUSPENSION CULTURE: ANALYSIS BY RAPID KINETIC TECHNIQUES (Proceedings Abstract). (Eng.) Marz, R. (Dept. Microbiology, Univ. Minnesota, Minneapolis, MN 55455); Wohlhueter, R. M. *Fed Proc* 35(7): 1759; 1976.

See also:

* (Rev): 3001, 3002, 3003, 3005, 3006, 3007, 3009, 3010, 3011, 3012, 3013, 3015, 3016, 3017, 3019, 3021, 3023, 3027, 3028, 3029, 3045, 3045, 3047, 3051, 3052, 3058
* (Chem): 3067, 3099, 3108
* (Phys): 3144
* (Viral): 3200, 3311, 3314
* (Immun): 3386
* (Path): 3400

3452 CHARACTERIZATION OF AN ESTABLISHED CELL LINE (SH-3) DERIVED FROM PLEURAL EFFUSION OF PATIENT WITH BREAST CANCER. (Eng.) Seman, G. (M. D. Anderson Hosp. and Tumor Inst., Texas Medical Center, Houston, TX 77025); Hunter, S. J.; Miller, R. C.; Dmochowski, L. *Cancer* 37(4): 1814-1824; 1976.

Spontaneous emergence of fast-growing cells in prolonged culture of pleural effusion cells obtained from a patient with breast cancer led to the establishment of a cell line designated as SH-3. Light and electron microscopy revealed that SH-3 cells are epithelial and resemble the poorly differentiated cells of a series of established human tumor cell lines. Their karyotype is hypotetraploid and different from that of HeLa cells. Isoenzyme analysis revealed the presence in the cells of a bone-type alkaline phosphatase, and of the fast (A) band of glucose-6-phosphate dehydrogenase. The line was free of mycoplasma.

3453 IN VITRO CULTURE TECHNIQUES. (Eng.) Klein, E. (Karolinska Inst., Stockholm, Sweden). *Int Union Cancer Tech Rep Ser* 20: 110-113; 1975.

In vitro cell culture techniques are discussed, including cloning of cells, preparation of monolayer cultures from solid tissue, and culturing of hematopoietic cell lines. Clean glassware is the basis for success in growing tissue cultures, where contamination with toxic substances and bacteria are the greatest hazards. Culture dishes must be in a CO_2 incubator where a mixture of 3%-5% CO_2 in air flows continuously. Cell strains will remain viable after freezing and storage at -70 C in a mixture of dimethyl sulfoxide, 10%, plus calf serum, 15%, in Eagle's medium. The isolation of cell lines by cloning is described. Two procedures for preparing monolayer cultures from solid tissue are: (a) 1-2 mm diameter tissue fragments are allowed to settle on the surface of the glass, where outgrowth takes place, and a confluent layer is obtained; (b) a tumor piece is broken up by trypsinization and centrifugation, and the cells are counted in a hemocytometer and suspended in growth medium (10^5 cells/ml) before distribution in culture flasks. Conventional methods have not been very successful in culturing hematopoietic cell lines unless impractically large quantities of lymphocytes are used. The gelatin foam (Spongostan) grid culture technique, however, provides an improvement for culturing lymphocytes and has also been effective with biopsies of nasopharyngeal carcinoma, Burkitt's lymphoma, normal and myeloma- or leukemia-infiltrated bone marrows, and spleens.

3454 NON-LYTIC, NON-IONIC DETERGENT EXTRACTION OF PLASMA MEMBRANE CONSTITUENTS FROM NORMAL AND TRANSFORMED FIBROBLASTS. (Eng.) Pearlstein, E. (New York Univ. Medical Center, 400 East 34 St., New York, NY 10016); Seaver, J. *Biochim Biophys Acta* 426(4): 589-597; 1976.

A technique to extract plasma membrane protein constituents selectively from normal and transformed cells that uses sublytic concentrations of nonionic detergents is presented. The procedure does not damage cells, as judged by cell viability, ^{51}Cr release, and trypan blue staining. Lactoperoxidase-catalyzed iodination followed by detergent extraction permits demonstration of a 100,000-dalton protein that is found on the surface of normal but not transformed hamster and mouse fibroblasts.

3455 MULTIDIMENSIONAL RESTRICTION ENZYME ANALYSIS OF COMPLEX GENOMES. (Eng.) Potter, S. S. (Sch. Medicine Univ. North Carolina, Chapel Hill, NH 27514); Newbold, J. W. *Anal Biochem* 71(2): 452-458; 1976.

The procedure that allowed the specific cleavage of *Escherichia coli* DNA and resolution of the resultant DNA segments into discrete bands on agarose gels is described. The method first involves DNA cleavage with one restriction enzyme (*Eco*Rl) followed by fractionation of the resulting DNA segments according to size using electroelution. The DNA in each fraction is then digested with another restriction enzyme (*Sma*l) and analyzed on a slab gel. Even more complex genomes could be analyzed by adding another stage of digestion with another restriction enzyme and electroelution. Possible applications include study of tandem repeats, analysis of subtle differences between closely related genomes, and purification of specific genes.

3456 QUANTITATIVE DETERMINATION OF POLY(ADENOSINE DIPHOSPHATE RIBOSE) IN DIFFERENT HEPATIC TISSUES BY AN ISOTOPE DILUTION PROCEDURE. (Eng.) Stone, P. R. (Natl. Cancer Inst., Natl. Insts. Health, Bethesda, MD 20014); Bredehorst, R.; Kittler, M.; Lengyel, H.; Hilz, H. *Hoppe Seylers Z Physiol Chem* 357(1): 51-56; 1976.

A procedure was developed to quantitate poly(ADP ribose) in intact hepatic tissues with different proliferation rates. It is based on the dilution of added ^{3}H-poly(ADP ribose) by the endogenous polymer. Five to 6 nmol of protein-bound ADP ribose per milligram of DNA were found in adult and neonatal rat liver, while Zajdela hepatoma cells had significantly lower values. This may reflect a true difference of the malignant cells, or may result from degradation of endogenous polymer during the washing procedure used to free the hepatoma cells from RBC. The similar levels of monomeric and polymeric ADP ribose residues in adult rat liver indicated that more proteins (or acceptor sites on proteins) must be occupied by single ADP ribose residues than by oligo- or poly(ADP ribose) chains. The mono (ADP ribose) levels of the different tissues correlated with the corresponding proliferation rates, being low in rapidly growing tissues, while the poly(ADP ribose) levels did not. These data indicate that the number of proteins, or the number of acceptor sites in proteins modified by ADP ribosylation, is determined by mono(ADP ribose) residues.

3457 ISOLATION OF A GOLGI-APPARATUS-ENRICHED FRACTION FROM LEUKAEMIC CELLS. (Eng.)

Warley, A. (Strangeways Res. Lab., Wort's Causeway, Cambridge CB1 4RN, United Kingdom); Cook, G. M. W. *Biochem J* 156(2): 245-251; 1976.

A method for isolating enriched fractions in the Golgi apparatus from lymphoblastic leukemic cells of AKR mice is presented. The fraction was isolated by a homogenate stabilized with 1 mM glutaraldehyde. The isolated fraction, which was shown morphologically to be enriched in dictyosomes, possessed a 44- to 76-fold increase in specific activity, compared with the tumor homogenate, of uridine 5'-diphosphate (UDP)-galactose-glycoprotein galactosyltransferase and a 3- to 10.5-fold increase in relative specific activity of UDP-*N*-acetylgalactosamine-polypeptide-*N*-acetylgalactosaminyltransferase. Plasma membranes isolated from the leukemic lymphoblasts also possessed glycoprotein galactosyltransferase activity, though in contrast with Golgi-apparatus-enriched material, they had no detectable polypeptide *N*-acetylgalactosaminyltransferase. The production of a Golgi-apparatus enriched fraction from leukemic cells may be useful to elucidate further the role of this organelle in membrane biogenesis and the control of enzyme expression in the plasma membrane.

3458 CYTOMETRIC ANALYSIS OF NEOPLASTIC TRANSFORMATION OF VERTEBRATE CELL POPULATIONS.

(Eng.) Fox, C. H. (Natl. Cancer Inst., Natl. Inst. Health, Bethesda, MD 20014); Dvorak, J. A.; Sanford, K. K. *Cancer Res* 36(5): 1556-1561; 1976.

Two parameters of neoplastic transformation in spontaneously transforming fibroblasts were examined. One parameter, the predilection for rounding of cells at metaphase, was studied using time-lapse cinephotomicrography and fixed, stained preparations. Ten different cell lines were assayed, including established lines of hamster, rat, and mouse cells, and six rodent cultures of known neoplastic potential as determined by animal injection and tumor production. There was a close correlation between the rounding of cells at metaphase and their ability to form tumors on im or intraocular injection in syngeneic hosts. The projected area of adherent cells 24 hr after plating over a coverglass was used to assay the transition of these rodent cell populations in culture from nonneoplastic to neoplastic. As the cell population became neoplastic, there was a significant decrease in the mean projected area of the cells (the second parameter). As the cell cultures became capable of producing tumors, the projected area profile of the population shifted proportionately to smaller area classes.

3459 KINETICS OF CELL PROLIFERATION IN NORMAL ENDOMETRIAL TISSUE AND IN ADENOCARCINOMA OF THE UTERUS: IMPULSE CYTOPHOTOMETRIC STUDY. (Dut.)

van Lindert, A. C. M. (No affiliation given); Vooijs, G. P.; Eskes, T. K. A. B. *Ned Tijdschr Geneeskd* 119(49): 1971-1972; 1975.

Pulse cytophotometry in combination with ethidium bromide as the DNA fluorescing material is a great asset in the study of the kinetics of cell proliferation. The instrument used was automated and could measure up to 1,000 cell nuclei per second. Accordingly, endometrial tissue obtained by curettage was dispersed as single cells in a trypsin medium. The suspension was first digested in pepsin to break down the cytoplasm and then colored with ethidium bromide. This method shows the differences in the S and G_2 + M phases clearly and thus is a valuable tool for the diagnosis and followup of treatment of adenocarcinoma of the uterus. (No references)

3460 APPARENT INHIBITORY INFLUENCE OF STRESS ON SJL/JDG NEOPLASIA.

(Eng.) Crispens, C. G., Jr. (Univ. Alabama in Birmingham, Birmingham, AL 35294). *IRCS Med Sci* 4(4): 169; 1976.

Weanling male SJL/JDg mice were caged singly and collectively (three per cage), and the incidence of spontaneous type B reticulum cell sarcomas was studied. Females (four per cage) were studied for comparison. The adrenal weights for singly caged males and for the females were normal, but the adrenals of the collectively caged males were larger than those of the singly caged males and showed a progressive increase in weight. The incidence of the sarcomas was significantly lower in the collectively caged males (6% and 28% at 30 and 52 wk, respectively) than in the singly caged males (40% and 63%, respectively). The respective values in the females were 64% and 86%. It is concluded that stress has an inhibitory effect on the development of these sarcomas in this mouse strain.

3461 SOLID TUMOR MODELS FOR THE ASSESSMENT OF DIFFERENT TREATMENT MODALITIES: II: RAPID, INTERMEDIATE, AND SLOW GROWING TRANSPLANTABLE RAT HEPATOMAS.

(Eng.) Looney, W. B. (Univ. Virginia Sch. Medicine, Charlottesville, VA 22901); Mayo, A. A.; Kovacs, C. J.; Hopkins, H. A.; Simon, R.; Morris, H. P. *Life Sci* 18(4): 377-389; 1976.

The effects of chemotherapeutic agents were studied at four organizational levels (molecular, cellular, organ, and animal) in three solid tumor models selected from a series of Morris hepatomas. Hepatoma H-4-II-E, which metastasizes to the lungs and axillary nodes, and the slow-growing hepatoma 16 were chosen for quantitative evaluation of the response to therapy in both tumor and host tissue. Hepatoma 3924A, a nonmetastasizing tumor, was selected for study of tumor effects on host immune responses and tumor response to immunotherapy alone or in combination with other modalities. Biophysical, biochemical, cell-kinetic, pathological, and immunological parameters were used to analyze changes following therapy. Biological markers used for the assessment of therapeutic response and surveillance for cancer recurrence included polyamine concentrations in both the tumor and serum of rapid, intermediate, and slow growing hepatoma-bearing animals. There was an inverse relationship between spermine concentrations and tumor growth rates. Major changes occurred in trace element concentrations in the blood, liver, and tumor in hepatoma 3924A-bearing animals following radiation treatment. Serum α-fetoprotein and cyclic AMP and cyclic guanosine 5'-monophosphate were also used as biochemical markers of tumor behavior. Temporal changes in individual tumor size were determined in the 10 different hepatoma lines that have volume doubling times ranging from 49 to 587 hr. The volume doubling times show a more than tenfold difference between the rapidly growing H-4-II-E and hepatoma 16. A twentyfold difference was observed between the 1-hr thymidine labeling index of the most rapid and the slowest growing tumor lines, suggesting a correlation between the proliferative compartment of the tumor, its actual growth rate, and its potential doubling time. These results indicate that each tumor line has specific cell-kinetic parameters that produce the net tumor growth rate. The three tumor lines selected permit evaluation of the major problems involved in attempts to improve the clinical management of solid tumors. (52 references)

3462 THE INFLUENCE OF A GRANULOCYTIC INHIBITOR(S) ON HEMATOPOIESIS IN AN IN VIVO CULTURE SYSTEM.

(Eng.) MacVittie, T. J. (Armed Forces Radiobiology Res. Inst., Defense Nuclear Agency, Bethesda, MD 20014); McCarthy, K. F. *Cell Tissue Kinet* 8(6): 553-559; 1975.

With the use of the in vivo diffusion chamber (DC) technique for mouse marrow culture, the effect of a granulocyte inhibitor on the proliferation of the pluripotent stem cell (CFU-s) and the granulocyte progenitor cell (CFU-c) was determined. Granulocyte conditioned medium was injected ip into mice bearing DCs during the initial 48 hr of culture. The early injection of inhibitor resulted in a significantly reduced number of granulocytic progeny formed within the DCs; there was no growth inhibition of mouse fibroblasts cultured under identical conditions. The reduced cell production was due partially to a significant reduction in the self-renewal rate of the CFU-c; no apparent direct effect was observed upon the growth of the CFU-s within the same cultures. The data suggested that the granulocytic inhibitor(s) acted to reduce proliferation within the CFU-c population and diminish the amplification potential inherent in the initial cell inoculum.

3463 ASSESSMENT OF TUMOR GROWTH.

(Eng.) Wagner, H. (Tiefenauspital, Bern, Switzerland). *Int Union Cancer Tech Rep Ser* 20: 44-51; 1975.

Analysis of tumor growth is possible if the variation of time of two of the following three parameters can be assessed: absolute tumor cell number, tumor cell production rate, and tumor cell loss rate. Since the absolute tumor cell number is often unknown, analyses of tumor growth are frequently based on estimates of relative tumor cell production and loss rates. Methods used for the assessment of cell production rates include the stathmokinetic agent plus mitotic index method, the double-labeling, the labeled mitoses, and the continuous labeling techniques. Direct measurements of cell death are not commonly used. Cell death in mitosis is difficult to assess because reliable morphologic criteria to distinguish dead from living cells in mitosis are not available and large numbers of mitoses would have to be screened to determine the relative number of dead cells. Cell death in interphase can be estimated by determining the relative number of non- or only partially-fragmented pyknoses. Procedures for in vivo and in vitro labeling of human bone marrow and blood cells and for radioautography (dipping, developing and fixing, and Giemsa staining) are outlined.

3464 EFFECT OF 6-HYDROXYDOPAMINE ON TUMOR GROWTH.

(Eng.) Chelmicka-Szorc, E. (Massachusetts General Hosp., Boston, MA 02114); Arnason, B. G. W. *Cancer Res* 36(7): 2382-2384; 1976.

Studies investigating the action of 6-hydroxydopamine (6-OH-DA), which exerts a neurotoxic action on catecholamine neurons, causing reversible damage to nerve endings in axotomized animals, and the possibility that loss of sympathetic function slows tumor growth are reported. The growth of C-1300 neuroblastoma was markedly slowed in 6-OH-DA-treated mice (6-wk-old male A/J and C57BL/6 strains). The growth of A-10 breast adenocarcinoma was also significantly retarded in 6-OH-DA-treated mice, but that of B-16 melanoma was not affected. In mice axotomized by pretreatment with 6-OH-DA, the growth of C-1300 neuroblastoma was slowed but the growth of the A-10 tumor was not affected. Results indicate that 6-OH-DA can act in two ways to slow tumor growth in vivo: by exerting a direct cytotoxic effect on tumor cells and by an indirect effect probably related to its ability to impede sympathetic function. An intact functional sympathetic nervous system may be a factor that determines the growth rate of certain tumors in vivo. (16 references)

3465 DETECTION OF EPIDERMAL GROWTH FACTOR (EGF)-BINDING SITES ON HELA AND CORNEAL EPITHELIAL CELLS.

(Eng.) Revoltella, R. (Lab. Cell Biology, C.N.R., Via Romagnosi 18A, 00196, Rome, Italy); Pediconi, M.; Amini, M.; Bosman, C.; Bertolini, L. *Eur J Cancer* 12(2): 151-157; 1976.

The ability of HeLa, murine C 1300 neuroblastoma, murine sarcoma S/180, murine sarcoma 2S/37, murine L-929, murine kidney, murine liver, murine splenic lymphocytes, and murine and rabbit corneal epithelial cells to bind epidermal growth factor (EGF)-coated SRBC was studied. The EGF-coated SRBC were incubated with the enucleated cells in suspension, and rosette formation was studied microscopically. The inhibition of rosette formation by various biological and chemical inhibitors was also investigated. HeLa cells incubated with EGF-coated SRBC formed stable rosettes, as did primary cultures of rabbit and mouse corneal epithelial cells incubated with EGF-coated SRBC. Normal spleen lymphocytes and kidney, liver, fibroblast, and sarcoma cells exhibited a limited ability to bind EGF, and the rosettes thus formed were unstable. The neuroblastoma cells formed rosettes with nerve growth factor (NGF)-coated SRBC, but not with EGC-coated SRBC. Rosette formation with HeLa cells was dependent on the number of EGF molecules present on the erythrocytes. Trypsin and free EGF, but not colchicine, vinblastine, cytochalasin-B, neuroaminidase, phospholipases, NGF serum albumin, normal gamma globulins, ferritin, cytochrome-c, or ribonuclease inhibited rosette formation by HeLa cells. Free NGF inhibited rosette formation by neuroblastoma cells. Even in the presence of effective inhibitor, the binding reaction reappeared within 2 hr. The binding of EGF to HeLa cells occurred at specific binding sites exposed on the cell-surface membranes of the cells. (17 references)

3466 POSSIBLE LIMITATION OF GROWTH IN HUMAN FIBROBLAST CULTURES BY DIFFUSION. (Eng.) Froehlich, J. E. (Dept. Medicine, Queen's Univ., Kingston, Ontario, Canada); Anastassiades, T. P. *J Cell Physiol* 86(3): 567-580; 1975.

The secondary cultures of human diploid fibroblasts that demonstrated density dependent inhibition of cell growth were used to study the possible limitation of growth in cell cultures by diffusion. An oscillating platform system was described that insured constant mixing of the medium during the culturing period. A greater number of cells in density inhibited cultures, grown to confluence for 4 days after initial seedling, could be stimulated to resume growth by a fresh medium change if the cultures were left undisturbed. This greater stimulation on the platform was probably not due to mechanical alterations on the surface of the cells due to motion of the medium. Despite the greater stimulation after a single treatment with fresh medium, refeeding the cells on the platform every other day over a 12-day period did not affect the final saturation density achieved in the cultures. Diffusion limitation of growth might occur under certain circumstances.

3467 POLYAMINE METABOLISM IN A RAT BRAIN TUMOR CELL LINE: ITS RELATIONSHIP TO THE GROWTH RATE. (Eng.) Heby, O. (Inst. Zoophysiology, Univ. Lund, Helgonavaegen 3, S-223 62 Lund, Sweden), Marton, L. J.; Wilson, C. B.; Martinez, H. M. *J Cell Physiol* 86(3): 511-522; 1975.

In order to determine whether the metabolism of the polyamines putrescine, spermidine, and spermine was related to cellular growth rate, the activities of L-ornithine decarboxylase and S-adenosyl-L-methionine decarboxylase as well as the levels of the polyamines in rat brain tumor cells at various stages of a 7-day in vitro growth period were measured and correlated with the continuous changes in specific growth rate. L-Ornithine decarboxylase and S-adenosyl-L-methionine decarboxylase both exhibited their maximal activities at the time of most rapid growth. A positive correlation between the activities of these enzymes and the specific growth rate of the tumor cells during the entire growth period was demonstrated. The pattern of fluctuation of the spermidine content during the culture cycle was similar to those of the enzyme activities and showed a high positive correlation with the specific growth rate of the tumor cells during the entire growth period. The putrescine content exhibited a low positive correlation, while the spermine content exhibited a somewhat higher (but negative) correlation with the specific growth rate. The high correlation between the specific growth rate of the tumor cells and the synthesis of the polyamines indicated that these events were primarily associated with processes involved in cell replication.

3468 POLYAMINE TRANSPORT AND METABOLISM IN MOUSE MAMMARY GLAND. (Eng.) Kano, K. (Natl. Inst. Arthritis, Metabolism, and Digestive Diseases, Natl. Inst. Health, Bethesda, MD 20014); Oka, T. *J Biol Chem* 251(9): 2795-2800; 1976.

The uptake system for spermidine by mouse (C_3H/HeN) mammary explants in culture is a time-dependent, energy-requiring process that can be stimulated by insulin and prolactin. The Vmax spermidine uptake, 1.90×10^{-4} μmol/mg/hr, was increased to 4.76×10^{-4} by insulin and to 7.24×10^{-4} μmol/mg/hr by insulin plus prolactin. Insulin prevented the efflux of sperimidine. Dinitrophenol, actinomycin D, and cycloheximide increased the spermidine efflux. Hydrocortisone alone or with insulin and/or prolactin did not stimulate uptake in a 2-day culture. The accumulated spermidine was virtually unchanged. In contrast, spermine and putrescine, both of which are also taken up by mammary explants, undergo metabolic conversion to spermidine.

3469 GROWTH RESPONSE TO LECTINS IN CHICK EMBRYO CELLS AT DIFFERENT STAGES OF DEVELOPMENT. (Eng.) Aubery, M. (Laboratoire de Biochimie, Faculte de Medecine Lariboisiere, Saint Louis Universite, Paris VII, 45, rue des Saints Peres, Paris 6e, France); Bourrillon, R. *Cell Differ* 5(1): 27-35; 1976.

The effect of Robinia lectin and Concanavalin A on fibroblasts and liver cells from chick embryos between the 8th and the 20th day of development was studied. In vitro after different times of cultivation. These lectins decreased cell number in cultures from young embryo cells but stimulated the proliferation of older embryo cells. The optimum concentration of either lectin was 3 μg/ml. No effect was observed on 12- and 14-day cells at the different concentrations of lectin used. The agglutination of fibroblasts by these lectins regularly decreased from the 8th to the 16th day of development. Liver cells, however, were agglutinated at no stage. These results could perhaps be explained in terms of cell surface changes either during the course of ontogeny or as a result of lectin treatment.

3470 MUTANTS OF BALB/C3T3 FIBROBLASTS DEFECTIVE IN ADHESIVENESS TO SUBSTRATUM: EVIDENCE FOR ALTERATION IN CELL SURFACE PROTEINS. (Eng.)

Pouyssegur, J. M. (Lab. Molecular Biology, Natl. Cancer Inst., Natl. Insts. Health, Bethesda, MD 20014); Pastan, I. *Proc Natl Acad Sci USA* 73(2): 544-548; 1976.

Two mutants of Balb/c 3T3 cells defective in adhesiveness to substratum were studied to investigate cell growth regulation mechanisms. After exposure to a mutagen a cell population was treated with prostaglandin E_1 and methylisobutylxanthine to elevate the cells 3':5'-cyclic AMP levels and adhesion to the substratum; then loosely adherent cells were harvested. After four cycles of selection two clones were isolated. Both were characterized by a round morphology and a decreased adhesion to substratum. The defect in adhesion appeared to be responsible for the change in cell shape, since the mutant cells were flattened and looked similar to wild-type cells when treated with dibutyryl cyclic AMP (though adhesiveness remains decreased). Lactoperoxidase-catalyzed iodination of the cell surface showed that two iodinated polypeptides (molecular wt, Mr, 90,000 and 135-140,000) present in wild-type cells not detected on the surface of the mutants, and another iodinated band (110-120,000 Mr) markedly decreased. Also, one mutant was missing the high-molecular-wt (230,000) cell surface protein that is sensitive to transformation. No changes were found in the content of cyclic AMP or free or polymerized tubulin. The exponential growth rate and saturation density was not altered in these adhesion-deficient cells. This last result suggests that modulation of adhesion to substratum per se does not play an important role in the growth control of Balb/c3T3 cells in vitro.

3471 MOLECULAR CHANGES IN CELL SURFACE MEMBRANES RESULTING FROM TRYPSINIZATION OF SARCOMA 180 TUMOR CELLS. (Eng.)

Huggins, J. W. (Dept. Biochemistry, Oklahoma State Univ., Stillwater, OK 74074); Chesnut, R. W.; Durham, N. N.; Carraway, K. L. *Biochim Biophys Acta* 426(4): 630-637; 1976.

Sarcoma-180 tumor cells in culture or grown as an ascites form in the CD-1 mouse were subjected to mild trypsinization procedures to study morphological and molecular changes resulting from proteolysis. The cells attached to a substratum became rounded within 20 min and most underwent cell division, but they did not detach from the substratum. Removal of trypsin permitted the cells to go back to their original spindle shape over an 8- to 20-hr period. Surface membranes were isolated from trypsinized ascites and cultured cells and subjected to dodecyl sulfate-acrylamide gel electrophoresis. Both cell types showed the same two kinds of changes in electrophoretic patterns. First, there was a loss of glycoproteins from both cell types, even though they showed different complements of cell surface glycoproteins. Second, there was a loss of high molecular wt polypeptides, which may play a role in membrane stabilization and cell shape. The results further implicate these polypeptides in the control of cell morphology and offer circumstantial evidence for transmembrane interactions of surface glycoproteins with high molecular wt polypeptides as a factor in controlling cell morphology.

3472 ALTERED SURFACE TOPOLOGY AND MEMBRANE FUNCTIONS OF RAT THYMOCYTES ELUTED FROM NYLON WOOL COLUMNS. (Eng.)

Kwock, L. (Tufts-New England Medical Center, 171 Harrison Ave., Boston, MA 02111); Lin, P. S.; Wallach, D. F. H. *Biochim Biophys Acta* 426(4): 669-674; 1976.

The surface topology, amino acid transport, and cyclic AMP content of thymocytes from Wistar-Lewis rats were studied before and after passage through columns of nylon wool. Following incubation of thymocytes with nylon wool at 37 C, the eluted cells showed an increase in the number of microvilli per cell and a concominant elongation of the microvilli (0.22 vs 1.15 μm). Cyclic AMP levels were lowered by 30%-50% in nylon wool-treated thymocytes. Nylon wool-treated cells showed an impaired Na^+-dependent amino acid transport system (2-aminoisobutyrate), whereas the Na^+-independent amino acid transport system (1-aminocyclopentane-1-carboxylate) was unaffected. The results indicate that caution must be applied when one uses columns to separate specific classes of lymphoid cells, as their surface morphology and amino acid transport are modified in the process.

3473 SYNTHESIS OF MICROTUBULE PROTEIN IN HELA CELLS APPROACHING DIVISION. (Eng.)

Lawrence, J. H. (Dept. Pathology, Univ. Medical Buildings, Foresterhill, Aberdeen AB9 2ZD, Scotland); Wheatley, D. N. *Cytobios* 13(50/55): 167-179; 1975.

HeLa cells at log phase growth in suspension culture were synchronized by amethopterin or hydroxyurea treatment for 14 hr. This procedure alone did not affect the specific binding activity of ^3H-colchicine to cytosol fractions prepared from 100,000 x g centrifugation of lysed cells. On release of cells arrested at the G_1/S boundary into S phase, a gradual increase in both relative and absolute amounts of microtubule protein (MTP) in the cell was observed; it reached a maximum coincident with the mitotic peak about 9 to 10 hr after reversal from the synchrony-inducing block. During the period of mid-S to M, the rise was not accompanied by large fluctuations. Results with a double synchrony technique were very similar. Mitotically active 3T6 cells in log phase growth had slightly higher ^3H-colchicine specific binding activity than cells which are almost entirely in G_1 stationary phase. A steady change in both absolute and relative MTP levels apparently occurs during the cell cycle, with the early part of the cycle being below average rising through S phase, with the latter part of the cycle being above average. MTP apparently does not have a regulatory function in the cell cycle, as mitosis is attempted with low MTP levels. The ability of cells to enter mitosis is divorced from their ability to develop spindles.

3474 ALPHA$_1$-ANTITRYPSIN AND ALPHA-FETOPROTEIN: PROTEIN MARKERS IN ENDODERMAL SINUS (YOLK SAC) TUMORS.

(Eng.) Palmer, P. E. (New England Medical Center Hosp., 171 Harrison Ave., Boston, MA 02111); Safaii, H.; Wolfe, H. J. *Am J Clin Pathol* 65(4): 575-582; 1976.

A combined immunocytochemical and quantitative serum and tissue study was performed on a group of endodermal sinus (yolk sac) tumors, localizing and measuring both α_1-antitrypsin (AAT) and α-fetoprotein (AFP) in tumor tissue and patient sera. By use of indirect immunofluorescent and triple-sandwich immunoperoxidase methods, both proteins were demonstrated within intra- and extracellular periodic acid-Schiff-positive hyaline globules characteristic of the tumor, as well as within the cytoplasm of tumor epithelial cells lining endodermal sinuses, where AAT deposition predominated. Tumor tissue extracts confirmed the presence of significant quantities of both proteins, and pretreatment serum elevations of both showed a parallel decline during therapy. In this study, AAT is characterized as a tumor protein marker for the first time, and a parallelism between AAT and AFP is demonstrated in both serum and tumor tissue. These findings represent evidence supporting the yolk sac origin of endodermal sinus tumors in man.

3475 IDENTIFICATION OF THE PRODUCTS OF MITOCHONDRIAL TRANSCRIPTION IN THE WALKER CARCINOSARCOMA BY THE USE OF ACTINOMYCIN D AND ETHIDIUM BROMIDE.

(Eng.) Gonzalez-Cadavid, N. F. (Departamento de Biologia Celular, Facultad de Ciencias, Universidad Central de Venezuela, Apartado 10098, Caracas, Venezuela); Perez, J. L. *Cancer Res* 36(5): 1754-1760; 1976.

The major RNA species in the purified mitochondrial fraction of the Walker carcinoma were studied to determine which of them are located in the mitochondria and coded by the organelle DNA. The subcellular distribution of these RNAs and the in vivo sensitivity of the transcription process to selective inhibitors were examined. Of the species separated by polyacrylamide gel electrophoresis, only the 21 and 16Se RNAs occurred exclusively in the purified mitochondria, Se being approximately the S value estimated from the relative electrophoretic mobility of the RNA. A bifid peak observed in the 16-15 Se region was shown to be an artifact caused by the ribonuclease inhibitor, naphthalene disulfonate. Ethidium bromide at high doses inhibited the incorporation in vivo of ^{32}P into 21, 16, and 4 Se RNA, but the nuclear transcription of cytoplasmic RNA was also inhibited to the same extent. No significant effect was observed at lower doses. In contrast, actinomycin D exerted a differential inhibition of the synthesis of 28 and 18 Se RNA from the cytoplasmic and the mitochondrial fractions, practically without affecting the transcription of the 21 and 16 Se species. The incorporation of ^{32}P into mitochondrial 4 Se RNA was also considerably more resistant to the drug than the synthesis of the cytoplasmic tRNA. It is concluded that the 21, 16, and 4 Se RNAs are the only major discrete species transcribed from mitochondrial DNA present in the Walker carcinoma.

3476 RADIOIMMUNOASSAY FOR SERUM THYROXINE-BINDING GLOBULIN: RESULTS IN NORMAL SUBJECTS AND IN PATIENTS WITH HEPATOCELLULAR CARCINOMA.

(Eng.) Gershengorn, M. C. (Natl. Inst. Arthritis, Metabolism, and Digestive Diseases, Natl. Insts. Health, Bethesda, MD 20014); Larsen, P. R.; Robbins, J. *J Clin Endocrinol Metab* 42(5): 907-911; 1976.

Serum thyroxine-binding globulin (TBG) was measured by radioimmunoassay and compared to the thyroxine (T_4)-binding capacity in groups of 98 and 45 normal subjects; the prevalence of elevated TBG was also determined in 19 hepatocellular carcinoma patients. The TBG used was purified by affinity, anion-exchange, and gel filtration chromatography. The serum TBG concentration in 98 euthyroid normals was 1.48 ± 0.46 mg/100 ml (mean ± SD), one-half the concentration previously reported using a similar method. The level in females (1.66 ± 0.56) was significantly higher than that in males (1.37 ± 0.37). Comparison of the serum TBG level and the maximum binding capacity of serum TBG for T_4 yielded a molar ratio of 1:1 for T_4 and TBG. The mean serum TBG in 19 hepatocellular carcinoma patients was 2.10 ± 1.29 mg/100 ml; however, only 2 had serum TBG levels outside the normal range. From these results it appears that elevated TBG is not a consistent finding in patients with hepatocellular carcinoma and that TBG is not a ''tumor marker'' for this neoplasm.

3477 EFFECT OF THE HEWITT KERATINIZING EPIDERMAL CARCINOMA ON CELL PROLIFERATION IN DIFFERENT ORGANS OF THE HOST MOUSE AND IN HUMAN PSORIATIC SKIN CULTURED IN DIFFUSION CHAMBERS.

(Eng.) Kariniemi, A.-L. (Second Dept. Pathology, Univ. Helsinki, Finland); Rytomaa, T. *Br J Dermatol* 94(5): 515-522; 1976.

Incorporation of ^3H-thymidine into different squamous epithelia and other tissues was studied in WHT/Ht mice bearing the Hewitt keratinizing epidermal carcinoma with a raised epidermal chalone content in the body. DNA synthesis in the tumor-bearing mice was inhbited 80% in the epithelium of the ear, 55% in the epithelium of the back skin and the foot, and 25% in the epithelium of the tongue and the cornea. Cell proliferation was not detectably affected in the sebaceous gland, intestinal epithelium, bone marrow, or thymus lymphocytes. Thymus size was reduced in the tumor-bearing mice compared with controls, and ^3H-thymidine uptake was depressed in the spleen and to some extent also in the lymph nodes. Labeling index of human psoriatic epidermis was suppressed 22% in diffusion-chamber cultures carried by the tumor-bearing host mice compared with controls. Psoriatic explants were used because they display more uniform labeling of epidermal cells in diffusion chamber cultures than normal skin explants do. This reaction was probably not specific, because the labeling index of mouse bone-marrow cells was also decreased when grown in tumor-bearing animals. These results are consistent with release of epidermal chalone by the Hewitt keratinizing epidermal carcinoma.

3478 MEASUREMENT OF FIBRINOGEN DEGRADATION PRODUCTS (FDP) IN SERUM OF NORMAL AND TUMOR-BEARING RATS. (Eng.) Abe, K. (Sch. Medicine, Tohoku Univ. Sendai, Japan); Fujimoto, H.; Endo, E.; Fukuoka, Y.; Khato, J.; Sato, H. *Tohoku J Exp Med* 117(4): 343-350; 1976.

In order to analyze the mechanism of fibrinolysis under tumor-bearing conditions, the level of serum fibrinogen degradation products (FDP) in Donryu rats was quantified by hemagglutination-inhibition immunoassay, which utilized the anti-rat-fibrinogen antiserum of rabbit and fibrinogen-coated rat erythrocytes. The normal value of serum FDP ranged from 1.25 to 10 μg/ml, with a mean value of 5.7 μg/ml. The changes in FDP level were noted in tumor-bearing rats that were transplanted subcutaneously with 10^6 tumor cells (AH-109A, a strain of Yoshida rat ascites hepatoma). The FDP level was unchanged in the early stages, rose to 56 μg/ml on the 12th day, and rose to 92 μg/ml on the 16th day, on an average. These elevations were statistically significant.

3479 EFFECT OF PROTEIN DEPRIVATION AND A REDUCED DIET ON THE REGENERATING RAT LIVER. (Eng.) Stirling, G. A. (Univ. Nottingham Medical Sch., Nottingham, England); Bourne, L. D.; Marsh, T. *Br J Exp Pathol* 56(6): 502-509; 1975.

Wistar rats fed a protein-free, calorie rich diet showed reduced DNA synthesis, diminished mitotic index, and impaired proliferative response. Nevertheless, protein-deficient hepatocytes retained a remarkable ability to hypertrophy and proliferate after partial hepatectomy. In pair-fed rats on a reduced semistarvation diet deficient in calories, these same parameters were not only diminished but their onset was also delayed. Electron microscopy revealed that the glycogen in the liver cells was severely depleted. This was considered to be a critical factor in the delayed response to partial hepatectomy. Both groups showed a marked reduction of organelles, especially ribosomes and rough endoplasmic reticulum. Golgi complexes persisted and were conspicuous. Rough endoplasmic reticulum was promptly reformed following partial hepatectomy.

3480 MEASUREMENT OF DNA CONTENT AND CELL VOLUME IN SENESCENT HUMAN FIBROBLASTS UTILIZING FLOW MULTIPARAMETER SINGLE CELL ANALYSIS. (Eng.) Schneider, E. L. (Natl. Inst. Aging, Bethesda, MD 20014); Fowlkes, B. J. *Exp Cell Res* 98(2): 298-302; 1976.

Cell cycle analysis of senescent cultured WI38 human fibroblasts by flow cytofluorometry revealed an increased proportion of cells in the G1 period. Both increased variation and slight decreases in cellular DNA contents were observed in both G1 and G2 + M senescent cells. With the use of gated single parameter analysis, increased cell volumes in these senescent fibroblasts were also observed. Senescent cells apparently have increased cell size in both G1 and G2 + M in logarithmic growth and confluency. Perhaps a cell membrane alteration leads to a loss in replicative ability, and the cell volume increases.

3481 DIURNAL RHYTHM OF MITOTIC ACTIVITY AND OF NUMBER OF DNA-SYNTHESIZING NUCLEI IN SARCOMA 37 OF ALBINO MICE. (Eng.) Demskii, V. I. (N. I. Pirogov Second Moscow Medical Inst., Moscow, USSR). *Bull Exp Biol Med* 80(10): 1244-1245; 1975.

Male albino mice with ascites tumor cells of sarcoma 37 were killed 1.0-1.5 hr after injection of ^{3}H-thymidine. The mitotic index in the tumor was maximal at 10:00 a.m. and minimal at 1:00 a.m. The differences in the diurnal rhythms of the index of labeled mitoses and the index of unlabeled mitoses indicated the existence of two cell populations. The G_2 phase in one group was about 1 hr in duration and that of the other group was about 4 hr or more.

3482 NATURAL SYNCHRONY OF NEWBORN MOUSE EPIDERMAL CELLS IN VITRO. (Eng.) Elgjo, K. (Div. Cancer Cause and Prevention, Natl. Cancer Inst., Bethesda, MD 20014); Hennings, H.; Michael, D.; Yuspa, S. H. *J Invest Dermatol* 66(5): 292-296; 1976.

DNA synthesis and mitotic rate were examined every few hours for the first 96 hr of in vitro growth of newborn mouse (BALB/c) epidermal cells. Cell proliferation was examined at short intervals during the first 3 to 4 days in culture; DNA synthesis was estimated by incorporating ^{3}H-thymidine into epidermal DNA or by the labeling index. The mitotic rate was estimated by blocking mitotic cells with vinblastine. Reasonable agreement was found with the different methods. The cells grew synchronously with peaks of DNA synthesis at 21 to 23 hr, 35 to 40 hr, and 60 hr; peak mitotic rates were seen at 44 to 48 hr and 72 to 76 hr. The cells appeared to grow exponentially during the first 3 to 4 days in culture, with one main cohort of cells dividing during the successive proliferative peaks. The presence of such synchrony may expedite the study of the effects of chemical compounds.

3483 INFREQUENT CHROMOSOME ABERRATIONS IN SYRIAN HAMSTER CELLS FOLLOWING PARTIAL SYNCHRONY BY AMINO-ACID DEPRIVATION. (Eng.) Popescu, N. C. (Natl. Cancer Inst., Building 37, Room 2A13, Bethesda, Md. 20014); Casto, B. C.; DiPaolo*, J. A. *J Cell Physiol* 86(3): 599-604; 1975.

The incubation of Syrian hamster embryo secondary cell cultures in isoleucine or arginine deficient medium inhibited cell multiplication; a total of 4-7% of the cells were synthesizing DNA compared to approximately 40% for cells in complete medium.

After the deficient medium was replaced with complete medium, cells resumed multiplication; within 12 hr 65-70% of the cells entered into the S phase. A second peak of labeled cells, approximately 55%, occurred 24 hr after addition of complete medium. Chromosomal damage characteristic for cell lines grown in amino acid deficient medium did not occur with these Syrian hamster cells. Isoleucine or arginine deprivation resulted in chromosome or chromatid aberrations in only 2-5% of the cells; chromosomes of control cells in complete medium occasionally exhibited chromatid gaps.

3484 THE BINDING OF TRITIATED ELONGATION-FACTORS 1 AND 2 TO RIBOSOMES FROM KREBS II MOUSE ASCITES-TUMOR CELLS. THE INFLUENCE OF VARIOUS ANTIBIOTICS AND TOXINS. (Eng.) Noland, R. D. (Sandoz Forschungsinstitut, GmbH, Brunnerstrasse 59, A-1235 Wien, Austria); Grasmuk, H.; Drews, J. *Eur J Biochem* 64(1): 69-75; 1976.

The effect of a number of different antibiotics and toxins on the capacity of Krebs II mouse ascites ribosomes to bind ^{3}H-labeled elongation factors (EF-1 and EF-2) was examined. It was found that abrin and ricin inhibited the binding of EF-2, while diphtheria toxin, sparsomycin, streptovitacin A, and cycloheximide had essentially no effect on its binding. These results suggest that cycloheximide functions by uncoupling the ribosome binding and translocation functions of EF-2. Of the other compounds examined, sparsomycin was unique in its capacity, under some circumstances, to affect significantly the binding of aminoacyl-tRNA and EF-1 to ribosomes. Fusidic acid appeared to enhance the binding of both EF-1 and EF-2 nonspecifically.

3485 THYMINE DIMER EXCISION BY EXTRACTS OF HUMAN CELLS. (Eng.) Duncan, J.; Slor, H.; Cook, K.; Friedberg, E. C. In: *Molecular Mechanisms for Repair of DNA, Part B.* Edited by P. C. Hanawalt and R. B. Setlow. (New York: Plenum Press): pp. 643-649; 1975.

Thymine dimer excision was studied with the use of human cells in tissue culture and cell-free extracts. The cells used were WIL II, a human lymphocyte line that grows in suspension culture; HeLa, originally derived from a human cervical cancer; WI-38, a human diploid fibroblast strain; or A-148, a human amnion-derived fibroblast line. In tissue culture experiments, after UV irradiation at 200 ergs/mm^2, 50% of the thymine dimers were excised in 4 hr, after which little if any loss of dimers from acid-precipitable DNA was observed. In cell-free extracts, the extent of dimer excision was about the same as in intact cells. Preirradiation of cells was not required for excision of thymine dimers; qualitatively similar results were obtained with extracts of unlabeled cells with use of ^{3}H-labeled UV-irradiated T7 DNA as the substrate. Thymine dimers were not excised by cell-free extracts if the cells were frozen prior to disruption or if the cell-free extracts were frozen. When UV-irradiated T7 DNA previously incised by T4 UV endonuclease was incubated with extracts of WIL II cells, excision reached a maximum of 50% within 20 min, at which time only about 5% of the total DNA was acid-soluble. This activity was detected in extracts of all cell lines and strains examined. Preliminary characterization of crude extracts indicated the following: (a) the dimer-excising activity is completely inactivated by SH-group inhibitors; (b) it has an absolute requirement for added divalent cation in the form of either $MgCl_2$ or $MnCl_2$ and is completely destroyed by EDTA; (c) it has a broad pH optimum, 6.8-8.3; and (d) it can work in the 5'→3' direction. Extracts of human cells grown in tissue culture are concluded to contain both UV endonuclease and dimer-excision activities.

3486 ON THE HETEROGENEITY OF NATIVE RIBOSOMAL SUBUNITS IN EHRLICH-ASCITES-TUMOR CELLS CULTURED IN VITRO. (Eng.) Van Venrooij, W. J. (Dept. Biochemistry, Univ. Nijmegen, Geert Grooteplein Noord 21, Nijmegen, The Netherlands); Janssen, A. P. M.; Hoeymakers, J. H.; De Man, B. M. *Eur J Biochem* 64(2): 429-435; 1976.

Analysis by high resolution CsCl isopycnic centrifugation of 'native' small ribosomal sub-units (40S) in cultured Ehrlich ascites tumor cells reveal at least five classes of particles. These particles, with buoyant densities of 1.39, 1.42, 1.45, 1.49, and 1.51 g/cm^3 (designated SI, SII, SIII, SIV, and SV, respectively), were different from the ribosome-derived 40S subunits that have a density of 1.54 g/cm^3. 'Native' large ribosomal subunits (60S) consist of at least two classes of particles with densities of 1.57 g/cm^3 (LI) and 1.59 g/cm^3 (LII), respectively. The ribosome-derived 60S subunits had the same buoyant density as the LII particles. Labeling of Ehrlich ascites cells with radioactive uridine and a subsequent chase in the presence of RNA-synthesis inhibitors showed that radioactivity was incorporated first in precursor particles with a density of 1.48 g/cm^3 and then subsequently appeared in SIII and SII particles. Met-tRNAf was found exclusively on 'native' 40S particles with densities of 1.42 and 1.49 g/cm^3. This result has been observed in cells labeled with [^{35}S]methionine in vivo as well as with tRNA labeled in vitro.

3487 COMPARISON BETWEEN THE RIBOSOMAL RIBONUCLEIC ACIDS FROM FREE AND MEMBRANE-BOUND RIBOSOMAL FRACTIONS OF HELA CELLS. (Eng.) Khan, M. S. N. (Dept. Biochemistry, Univ. Glasgow, Glasgow G12 8QQ, U.K.); Maden, B. E. H. *Biochem J* 155(1): 197-200; 1976.

The ribosomal RNA (rRNA) species from the total cytoplasmic, free, and membrane-bound fractions of HeLa cells were compared, to determine a basis for functional differentiation between classes of ribosomes. Using T_1 ribonuclease and combined T_1 ribonuclease plus pancreatic ribonuclease fingerprinting procedures, it was found that no significant differences between the rRNA species from the different subcellular fractions. The results are consistent with models for ribosome function that envisage exchange of ribosomal subunits between free and membrane-bound situations, possibly as a result of the particular polypeptide being synthesized.

3488 METHYLATED, BLOCKED 5' TERMINI OF YEAST mRNA.

(Eng.) Sripati, C. E. (Albert Einstein Coll. Medicine, Bronx, New York NY 10461); Groner, Y.; Warner, J. R. *J Biol Chem* 251(10): 2898-2904; 1976.

mRNAs of the yeast, *Saccharomyces cerevisiae*, strain A 364A, have modified 5' termini similar but not identical with those present in the higher eukaryotes; this study was undertaken in the hopes of using this yeast to study mRNA synthesis. Poly(A)-containing RNA isolated from spheroplasts or from polysomes of cells labeled with [methyl-^{3}H]methionine or $^{32}P_1$ contain methylated, blocked 5'-terminal structures of the composition m^{7}G(5')pppAP and m^{7}G(5')pppGp occurring in a relative distribution of 75% and 25%, respectively. Furthermore, there is no N^{5}-methyladenosine nor any other methylated nucleoside in the entire molecule of poly(A)-containing RNA. Experiments with a mutant (mutation in gene rna 1) temperature-sensitive for the transport of mRNA to cytoplasm suggest that the methylation and addition of the blocking group take place in the nucleus and that the genetic lesion in the mutant does not affect these mechanisms. This modified 5' terminus is therefore likely to be a common feature of all eukaryotic mRNA.

3489 XC-CELL FUSION INDUCED BY MURINE PLASMOCYTOMA CELLS. III. RNA GENE EXPRESSION CORRELATED TO SYNCYTIUM FORMATION.

(Eng.) Herisse, J. (Unite de Recherche de Virologie no 102 de l'I.N.S.E.R.M., 2, place de Verdun, 59045 Lille Cedex, France); Lemay, P.; Tavitian, A. *Biomedicine* [*Express*] 25(1): 3-7; 1976.

The viral gene expression in the MF$_2$ cell line (mouse myeloma-derived) cultured in vitro and in vivo was studied. The cells produced C-type viral particles when maintained in tissue culture. When cultured in an ascitic form by injection in Balb/c mice, they lost this property. The ability to induce syncytia by cocultivation of the MF$_2$ cell with XC-cells was related to the viral production. A DNA complementary to viral 70S RNA was synthesized by use of the viral reverse-transcriptase endogenous activity. Expression of the viral genome among cellular polyA-rich RNA varied concomitantly with the syncytium-inducing ability, as evidenced by molecular hybridization experiments.

3490 COLLAGENOLYTIC ENZYME IN SOLID TAWA SARCOMA.

(Eng.) Sakaki, T. (Osaka Dental Univ., 47, Kyobashi 1-chome, Higashi-ku, Osaka 540, Japan); Fujita, A.; Wakumoto, N.; Murase, S. *Gann* 67(1): 67-73; 1976.

In order to demonstrate a collagenolytic enzyme in a tumor, solid Tawa sarcoma was subjected to a tissue culture technique. Salt- and acid-soluble collagen used as substrate was extracted from Donryu rat skin and tail tendon. The tumor mass was obtained on the 5th, 8th, and 11th days after sc transplantation of ascites Tawa sarcoma cells. Each tissue fragment in the inner and outer layers of the tumor mass was incubated on collagen gel at 37 C for 4 days, and collagenolytic activity was determined by comparsion of the relative content of hydroxyproline in the attacked collagen, which was separated by means of centrifugation. All fragments were found to possess collagenolytic activity with some variation. Higher activity was observed in the outer layer than in the inner layer. Tumor fragments were cultured, and the collagenolytic enzyme was isolated from the culture medium and concentrated by ammonium sulfate precipitation and acrylamide disc electrophoresis. Electrophoresis of the products of purified collagenolytic enzyme revealed that the collagenolytic activity in the tumor involves a hydrolytic cleavage of collagen within its helical region.

3491 DETECTION AND ESTIMATION OF VERY LOW RIBONUCLEASE ACTIVITIES IN BIOLOGICAL FLUIDS.

(Eng.) Egly, J. M. (Unite(44) de Recherches Fondamentales sur la Biochimie de la Cellule Cancereuse de l'INSERM, 11, rue Humann, 67085--Strasbourg, Cedex, France); Kempf, J. *FEBS Lett* 63(2): 250-254; 1976.

A new procedure for the detection and estimation of small quantities of ribonuclease (RNase) activity in biological fluids is described. Iodine-labeled RNA with high specific activity (33,000 cpm/μg), bound by 5' terminal phosphate to beaded agarose and thereby insolubilized, is incubated with the sample to be tested (in these experiments, solid transplantable plasma cell tumor RPC$_5$ grown on Balb/c mice). With this method, the presence of RNase activities at a concentration down to 10^{-8} μg/ml [expressed as pancreatic RNase (EC 3.1.4.22)] in the incubated sample can be detected.

3492 TRANSCRIPTION IN VITRO OF EHRLICH ASCITES TUMOR DNA AND CHROMATIN BY PURIFIED HOMOLOGOUS RNA POLYMERASE II (OR B).

(Eng.) Chan, J. Y. H. (Div. Cell and Molecular Biology, State Univ. New York at Buffalo, Buffalo, NY 14214); Loor, R.-M.; Wang, T. Y. *Arch Biochem Biophys* 173(2): 564-576; 1976.

An electrophoretically homogeneous RNA polymerase II (α-amanitin-sensitive) was purified from Ehrlich ascites tumor cells and used to study transcription of homologous DNA and chromatin. The general characteristics in the transcription of DNA and chromatin by the purified RNA polymerase were compared with respect to metal ion requirements and sensitivity toward spermine, rose bengal, and heparin. The fidelity of the tumor RNA polymerase in transcription was examined by comparison with that of *Micrococcus luteus* RNA polymerase. Only a limited portion of DNA and chromatin was transcribed by the tumor RNA polymerase, while the bacterial enzyme transcribed both templates extensively. By double-saturation of DNA or chromatin with the two enzymes as well as kinetic studies, it was shown that the tumor RNA polymerase, in contrast to the *M. luteus* RNA polymerase, transcribed selective DNA sequences.

3493 DNA-DIRECTED RNA POLYMERASE FROM HELA CELLS: ISOLATION, CHARACTERIZATION AND CELL-CYCLE DISTRIBUTION OF THREE ENZYMES.

(Eng.) Benecke, B. J. (Institut fuer Physiologische Chemie, Philipps-Universitaet, D-355 Marburg, Lahnberge, W. Germany); Seifart, K. H. *Biochim Biophys Acta* 414(1): 44-58; 1975.

The DNA-directed RNA polymerase was solubilized from total HeLa cells. Three distinct classes of the enzyme could be differentiated by their sensitivity toward alpha-amanitin. While form A was completely resistant to high concentrations (133 μg/ml) of this toxin, enzyme B was highly-sensitive and was completely inhibited by concentrations of 0.1 μg/ml. RNA polymerase C showed an intermediate behavior. Separation of the three individual enzymes was achieved by chromatography on DEAE-cellulose (to separate enzyme B from A and C) and DEAE-Sephadex (to separate polymerase A from C). All three RNA polymerases were subsequently purified by phosphocellulose chromatography (followed by sedimentation through glycerol gradients). Analysis of the purified enzymes by gel electrophoresis under denaturing conditions showed that the A enzyme consisted of five subunits with molecular wt of 185, 128, 65, 41 and 32 x 10^3. Polymerase B was composed of seven subunits in variable stoichiometry with molecular wt of 215, 175, 145, 123, 68, 43 and 31 x 10^3. The subunit structure of enzyme C was not clear.

3494 PLASMA MEMBRANE LOCALIZATION OF ALKALINE PHOSPHATASE IN HELA CELLS.

(Eng.) Lin, C.-W. (Tufts Univ. Sch. Medicine, Boston, MA 02111); Sasaki, M.; Orcutt, M. L.; Miyayama, H.; Singer, R. M. *J Histochem Cytochem* 24(5): 659-667; 1976.

The localization of alkaline phosphatase (AP) in HeLa cells (TCRC-1 and TCRC-2 sublines) was examined by electron microscopic histochemistry and subcellular fractionation. The electron microscopic histochemical results showed that in both sublines the major location of AP is in the plasma membrane. The enzyme reaction was occasionally observed in some of the dense body lysosomes. This result was supported by data from a subcellular fractionation study that showed that the microsomal fraction rich in plasma membrane fragments had the highest activity of AP. The distribution of this enzyme among the subcellular fractions closely paralleled that of the 5-nucleotidase, a plasma membrane marker enzyme. Characterization of the AP present in each subcellular fraction showed identical enzyme properties, which suggests that a single isoenzyme exists among fractions obtained from each cell line. The results, therefore, confirm the concept that plasma membrane is the major site of AP localization in HeLa cells. The absence of any enzyme reaction in the perimitochondrial space also indicates that the mitochondrial localization of the Regan isoenzyme reported in ovarian cancer may not be a common phenomenon in Regan-producing cancer cells.

3495 INTRANUCLEAR DISTRIBUTION OF MOUSE LIVER NUCLEAR DNA POLYMERASE.

(Eng.) Philippe, M. (Laboratoire de Biologie Cellulaire, Universite Paris-Val de Marne, 94010 Creteil, France); De Recondo, A.-M.; Chevaillier, P. *Exp Cell Res* 98(2): 424-428; 1976.

Adult mouse liver nuclei and their subfractions corresponding to heterochromatin, nucleoli, membranes, and euchromatin were studied for DNA-polymerase activity. The intact nuclei and the two heavy nuclear fractions contained rather low activity, while the two light fractions (membranes and euchromatin) had no activity at all. In the two heavy fractions, the activity was stimulated by β-mercaptoethanol and depressed by addition of p-hydroxymercuribenzoate and by omission of one or more nucleotides. A nuclease activity, detected in the intact nuclei, may also be present in the nuclear subfractions. DNA-polymerase activity was only slightly modified with the addition of exogenous native DNA, while the addition of denatured DNA caused a reduction in activity. The enzyme linked to the nuclear fractions can apparently interchange with an exogenous template; the enzyme finds the exogenous or endogenous native DNA is apparently a better template than the denatured DNA.

3496 FURTHER CHARACTERIZATION OF A MITOCHONDRIAL DNA POLYMERASE FROM HELA CELLS.

(Eng.) Radsak, K. (Hygiene Inst. Univ. Marburg, Pilgrimstein 2, 355 Marburg, W. Germany); Knopf, K.-W.; Weissbach, A. *Biochem Biophys Res Commun* 70(2): 559-566; 1976.

To further purify and characterize HeLa cell mitochondrial DNA polymerase, it was extracted with high salt concentrations (1 M) and Nonidet P 40 (0.2%). Subsequently, the enzyme was purified stepwise by DEAE-cellulose, phosphocellulose, hydroxyapatite-Ultrogel, and DNA-cellulose chromatography and preparative polyacrylamide gel electrophoresis. The purified enzyme had a molecular wt between 100,000 and 110,000 and was devoid of endonuclease activity. Sodium dodecyl sulfate polyacrylamide gel electrophoresis revealed two protein bands, suggesting that the mitochondrial DNA polymerase might consist of two subunits with molecular wt of 45,000 and 60,000.

3497 DEVELOPMENTAL PHASE-SPECIFIC ALKALINE PHOSPHATASE ISOENZYMES OF HUMAN PLACENTA AND THEIR OCCURRENCE IN HUMAN CANCER.

(Eng.) Fishman, L. (Tufts Univ. Sch. Medicine, Boston, MA 02111); Miyayama, H.; Driscoll, S. G.; Fishman, W. H. *Cancer Res* 36(7): 2268-2273; 1976.

Alkaline phosphatase electrophoretic patterns characteristic of three phases in early human trophoblast development are described. Phase 1 (6 to 10 wk) consisted entirely of two heat-sensitive, L-homoarginine-inhibited bands: the slower one possessed antigenic determinants of liver-bone-type alkaline phosphatase; the fast band lacked any of the known alkaline phosphatase antigenic determinants. Phase 2 pattern (11 to 13 wk) was a mixture of Phase 1 and Phase 3 isozyme components, the latter exhibiting two isozyme bands with the characteristics of term placental alkaline phosphatase. These three patterns of developmental phase-specific placental alkaline phosphatases corresponded in order to non-Regan isoenzyme (hypothesized as early placental), a mixture of Regan (term placental) and non-Regan isozymes, and Regan isoenzyme in human cancer tissues. The biochemical profile characteristic of trophoblast developmental Phase 1 alkaline phosphatase was expressed as 78.5% heat-sensitive inhibition (5 min at 65 C), 66.3% L-homoarginine inhibition, and 17.3% L-phenylalanine inhibition (n = 12). It was hypothesized that the alkaline phosphatase of human tumor tissues reflects the expression of placental genes corresponding to one or more phases of trophoblastic development. (18 references)

3498 $(Na^+ + K^+)$-ACTIVATED ATPase AND K^+-ACTIVATED P-NITROPHENYL-PHOSPHATASE ACTIVITIES OF THE NERVOUS SYSTEM CELLS IN TISSUE CULTURE. (Eng.) Stefanovic, V. (Centre de Neurochimie de CNRS, 11 Rue Humann, F-67085 Strasbourg-Cedex, France); Ciesielski-Treska, J.; Ledig, M.; Mandel, P. *Experientia* 31(7): 807-808; 1975.

The $(Na^+ + K^+)$-ATPase activity was studied in four mouse neuroblastoma clones, NN hamster astroblasts, C6 glioblastoma cells, and in astroblasts from the newborn rat brain in primary culture. The four clones had activities of 0.44-0.62 μmol pi/mg protein/hr. The C6 cells, NN cells, and astroblasts had values of 0.67, 0.89, and 2.33 μmol pi/mg protein/hr. K^+-activated, ouabain-inhibited p-nitrophenylphosphatase activity was also present in these lines, with values ranging from 0.12 to 0.28 μmole p-nitrophenol released/mg protein/hr. The results further supported the view that the $(Na^+ + K^+)$-ATPase and the K^+-activated p-nitrophenylphosphatase activities are the same enzyme system.

3499 REGULATION OF PURINE METABOLISM: ADENYLOSUCCINATE SYNTHETASE FROM NOVIKOFF ASCITES TUMOR CELLS. (Eng.) Clark, S. W. (Dept. Biochemistry, Rice Univ., Houston, TX 77001); Rudolph, F. B. *Biochim Biophys Acta* 437(1): 87-93; 1976.

The preparation of adenylosuccinate synthetase from Novikoff ascites tumor cells is described, and the properties of the enzyme are compared to those of the analogous enzyme isolated from normal liver. The Km values for aspartate, IMP, and GTP were 840, 12, and 12 μM, respectively, compared to normal liver values of 360, 30, and 15 μM. The higher value for aspartate is considered the most significant and may represent alterations in the control of AMP biosynthesis in tumor cells. The lower value for IMP may represent more efficient AMP synthesis. The antibiotic hadacidin competed with aspartate for the tumor enzyme with a KI of 1.5 μM, compared to a value of 6.3 μM with the liver enzyme, suggesting a severalfold preferential inhibition of the tumor enzyme. The KI for the feedback inhibitor AMP was 160 μM, compared to 47 μM for the normal liver enzyme. This reduced feedback inhibition might allow the tumor cell enzyme to operate at a higher flux than the liver enzyme, thus allowing tumor cells to be more efficient in purine synthesis and interconversion, but it might also be more sensitive to specific antitumor drugs. The apparent molecular wt of the tumor enzyme is 55,000-60,000, similar to the bacterial and liver enzyme. The liver enzyme was relatively stable for 4-5 days at pH 6.5-7.5, but the tumor enzyme was relatively less stable at all pH's studied. (16 references)

3500 KINETIC PROPERTIES OF ALCOHOL DEHYDROGENASE IN HEPATOCELLULAR CARCINOMA AND NORMAL TISSUES OF RAT. (Eng.) Cederbaum, A. I. (Mount Sinai Sch. Medicine, City Univ. New York, New York, NY 10029); Rubin, E. *Cancer Res* 36(7): 2274-2277; 1976.

The properties of alcohol dehydrogenase of a rat hepatocellular carcinoma (Becker H-252), a tumor of intermediate growth rate, are different from those of the liver enzyme, suggesting different isozymes. To determine the effects of the degree of differentiation, a fast-growing, poorly differentiated tumor, Morris hepatoma 7288ctc, and one that is well differentiated and of intermediate growth rate, Morris hepatoma 5123C, were studied. Alcohol dehydrogenase from 7288ctc had properties similar to those found with the Becker H-252 tumor, including a high Km for ethanol and acetaldehyde and the absence of substrate inhibition. By contrast, alcohol dehydrogenase from 5123C had properties similar to those of the liver enzyme. Thus, alcohol dehydrogenase is another example of an enzyme the composition of which changes with neoplastic dedifferentiation. Further studies are required to determine the factors responsible for the biochemical defect that occurs at the molecular level during carcinogenesis and whether the alcohol dehydrogenase isozymes in the Becker H-252 and Morris 7288ctc hepatomas are identical. A survey of several normal rat tissues revealed that only the stomach contains the unique alcohol dehydrogenase isozyme. (16 references)

3501 MULTIPLE MOLECULAR FORMS OF LACTATE DEHYDROGENASE AND GLUCOSE 6-PHOSPHATE DEHYDROGENASE IN NORMAL AND ABNORMAL HUMAN BREAST TISSUES. (Eng.) Hilf, R. (Univ. Rochester Sch. Medicine and Dentistry, Rochester, NY 14642); Rector, W. D.; Orlando, R. A. *Cancer* 37(4): 1825-1830; 1976.

Normal and abnormal breast samples of women were analyzed for multiple molecular forms of lactate dehydrogenase and glucose 6-phosphate dehydrogenase, by use of acrylamide disc gel electrophoresis and specific enzyme staining techniques for separation and quantitation. Infiltrating ductal carcinomas demonstrated a significant increase (three- to sixfold) in the proportion of LDH-5, compared to samples of normal breast, fibrocystic disease, and fibroadenoma, indicative of a shift toward the muscle-type lactate dehydrogenase in neoplasia. For glucose 6-phosphate dehydrogenase, carcinomas were found to contain increased proportions of the fastest migrating species, G6PD-I. Total enzyme activity per milligram of DNA was elevated in neoplastic tissues. Little or no alteration in isoenzyme profiles could be related to the menopausal status of the patient.

3502 DIAGNOSIS OF RENAL TUMOR BY GAMMA-GLUTAMYLTRANSPEPTIDASE. (EC 2.3.2.2).

(Eng.) Hautmann, R. (D-5100 Aachen, Morillenhang 9, W. Germany); Kisters, R.; Lutzeyer, W. *Urology* 7(1): 12-16; 1976.

A urinary enzyme pattern and kidney tissue pattern were investigated simultaneously in 117 urologic patients. Enzyme activities were measured in centrifuged, dialyzed urine samples obtained by ureteral catheterization, and in homogenized tissue samples taken immediately after nephrectomy. Lactate dehydrogenase (LDH) was increased 10-fold in the urine and was present in normal levels in the tissues of the renal tumor patients, while malate dehydrogenase (MDH) was diminished in the tissue and increased in the urine, and γ-glutamyl transpeptidase (γ-GT) was significantly reduced in both tissue and urine. LDH and MDH were increased in every other case of renal disorder studied, and γ-GT showed a highly significant increase in all urologic patients except those with tumor. The results indicate that determinations of LDH activity are useful for screening, but are of no diagnostic value. In contrast, a decrease in γ-GT activity in a urologic patient is of diagnostic value since it appears reliably and exclusively in renal tumor patients.

3503 SERUM AND HOST LIVER ACTIVITIES OF GLYCOSIDASES AND SIALYLTRANSFERASES IN ANIMALS BEARING TRANSPLANTABLE TUMORS.

(Eng.) Bosmann, H. B. (Roswell Park Memorial Inst., Buffalo, N.Y. 14263); Spataro, A. C.; Myers, M. W.; Bernacki*, R. J.; Hillman, M. J.; Caputi, S. E. *Res Commun Chem Pathol Pharmacol* 12(3): 499-512; 1975.

Rats bearing Reuber H-35 or Novikoff hepatomas and mice bearing L1210 or L5178Y murine leukemias exhibited elevated serum levels of fetuin: N-acetylneuraminic acid transferase activity. The serum transferase activity could be correlated with the growth rate of the tumor; in rats bearing the Novikoff hepatoma, activity was higher than in rats bearing the Reuber H-35 hepatoma. Higher transferase levels were also found in L1210 leukemic mice than in mice with the slower growing L5178Y leukemia. Serum from rats bearing Reuber H-35 hepatoma and mice bearing L1210 murine leukemia had elevated levels of alpha- and beta-glucosidase, alpha- and beta-galactosidase, beta-mannosidase, alpha- and beta-fucosidase, beta-N-acetylglucosaminidase, and acid phosphatase. Alpha-mannosidase, beta-N-acetylgalactosaminidase, and beta-xylosidase were not elevated. In rats bearing Reuber H-35 hepatoma, host liver levels of glycosidases, beta-glucuronidase, and acid phosphatase were elevated over the control and the hepatoma values. Tumors or various host tissues were though to release large quantities of enzymes into the serum.

3504 STUDIES ON THE REGULATION OF ORNITHINE DECARBOXYLASE ACTIVITY BY THE MICROTUBULES: THE EFFECT OF COLCHICINE AND VINBLASTINE.

(Eng.) Chen, K. (Dept. Pharmacology, Yale Univ., New Haven, CT 06510); Heller, J.; Canellakis, E. S. *Biochem Biophys Res Commun* 68(2): 401-408; 1976.

The effects of colchicine and vinblastine were studied on the regulation of ornithine decarboxylase (ODC) activity by cellular microtubules, using mouse L1210 leukemia cells. Dilution of stationary L1210 cultures resulted in an increase of ODC activity, the stimulation of ODC activity being completely inhibited by vinblastine or colchicine. Vinblastine produced a 50% inhibition of ODC activity at 8×10^{-8} M, whereas colchicine caused 50% inhibition at 7×10^{-7} M. Lumicolchicine, which has no effect on the microtubular system, had no effect on ODC activity. However, the enzyme was inhibited 50% by 7×10^{-6} M cytochalasin B and 20% by 10^{-5} ouabain. Disruption of the microtubule system was not enough to inhibit ODC activity, temperature-dependent steps being required between disruption of the microtubules and inhibition of ODC activity. The data strongly suggest that the regulation of L1210 ODC activity is related to the integrity of the cytoplasmic microtubules. This provides a model system that connects the perturbation of the membrane with a cytoplasmic enzymatic marker. (28 references)

3505 IN VITRO TRANSLATION AND ESTRADIOL-17β INDUCTION OF *XENOPUS LAEVIS* VITELLOGENIN MESSENGER RNA.

(Eng.) Shapiro, D. J. (Dept. Biochemistry, Univ. Illinois, Urban, IL 61801); Baker, H. J.; Stitt, D. T. *J Biol Chem* 251(10): 3105-3111; 1976.

In order to analyze the estrogen induction of vitellogenin synthesis and vitellogenin mRNA, estradiol-17β was administered to male *Xenopus laevis*. RNA extracted from livers directed the synthesis of the entire 200,000-dalton vitellogenin monomer in a cell-free protein synthesizing system derived from rabbit reticulocytes. Vitellogenin thus synthesized in vitro was isolated and quantitated by indirect immunoprecipitation and identified by comparison to authentic [^{14}C]vitellogenin. The in vitro product and [^{14}C]vitellogenin comigrated on electrophoresis in sodium dodecyl sulfate-polyacrylamide gels and they exhibited identical immunoprecipitation curves. *Xenopus laevis* vitellogenin messenger RNA had a sedimentation coefficient of approximately 30S in sucrose density gradients. It was purified approximately 60-fold from cell RNA by poly(U)-Sepharose chromatography and appeared to contain a polyadenylate sequence. Vitellogenin mRNA and vitellogenin synthesis in vivo could not be detected in unstimulated male *Xenopus laevis*. The relative rate of vitellogenin synthesis and the level of vitellogenin mRNA were determined at various times following the administration of estradiol-17β. Vitellogenin synthesis was maximal 12 days after estradiol-17β administration, when it comprised approximately 70% of the cell protein synthesis. This induction of high levels of vitellogenin synthesis did not require the appearance of a new cell type, but it is remarkable that it occurred in a type of tissue that exhibits a specialized and diverse genetic potential.

3506 DISSOCIATION OF THE MITOTIC SPINDLE IN OOCYTES EXPOSED TO GRISEOFULVIN AND VINBLASTINE. (Eng.) Malawista, S. E. (Yale Univ. Sch. Medicine, New Haven, CT 06510); Sato, H.; Creasey, W. A. *Exp Cell Res* 193-197; 1975.

The effects of griseofulvin and vinblastine on the mitotic apparatus (meiosis I) of starfish (*Pisaster ochraceous*) were studied. The ovaries were dissected out and induced to spawn, and the oocytes obtained were incubated with vinblastine at 1×10^{-5} M or griseofulvin at 2×10^{-6} M, 1×10^{-5} M or 2×10^{-5} M. The spindles were mass isolated without disturbance of their birefringence or initial size. With vinblastine and the two higher concentrations of griseofulvin, a linear decrease occurred until 6 min had elapsed, after which no further decrease in retardation of the isolated meiotic spindles, as measured with a polarizing microscope, occurred. The cellular uptake of ^3H-griseofulvin and ^3H-vinblastine was also determined, with the use of the same concentrations as before. Griseofulvin showed an initial rapid phase, complete in 1 to 2 min, followed by a slower increase probably representing intracellular binding. The 1-2 $\times 10^{-6}$ M concentrations rapidly produced constant intracellular drug levels, as well as mitotic spindles that were persistent as well as miniature.

3507 THE INFLUENCE OF INSULIN ON VARIOUS ENZYME ACTIVITIES IN HUMAN AND RAT HEPATOMA CELLS. (Eng.) Grimm, J. (Dept. Biochemistry, Univ. Witwatersrand, Johannesburg, South Africa). *Eur J Biochem* 64(1): 249-253; 1976.

The activities of a number of enzymes of cells from human and rat hepatomas were studied with respect to their response to insulin, and these changes were correlated with the metabolism of cyclic AMP. Incubation of human and rat hepatoma cells with insulin (1mU/10^6 cells) decreased their content of adenosine 3':5'-monophosphate by more than half after 1 hr and by about a quarter after 4 hr. The activities of the ATP-metabolizing enzymes, adenylate kinase and Mg^{2+}-adenosine triphosphatase were significantly increased by insulin within 1 hr and after 4 hr. The activity of succinate dehydrogenase and lactic dehydrogenase showed no change at either time interval. Insulin markedly stimulated glucose-6-phosphate dehydrogenase activity within 1 hr, but by 4 hr the increase was less apparent. Glutamate dehydrogenase activity, by contrast, was not increased by 1 hr but at 4 hr was elevated. It appears that these hepatoma lines do not possess adenyl cyclase and that insulin caused a decrease in the intracellular content of cyclic AMP. It is not known why the stimulatory effect of ATPase, adenylate kinase, and glucose-6-phosphate dehydrogenase and decrease in cyclic AMP content is less after 4 hr than 1 hr. It is possible that a significant destruction of insulin takes place during this period.

3508 CELL-FREE SYNTHESIS OF A PROLACTIN PRECURSOR DIRECTED BY mRNA FROM CULTURED RAT PITUITARY CELLS. (Eng.) Evans, G. A. (Dept. Medicine, Univ. California, San Diego, CA 92093); Rosenfeld, M. G. *J Biol Chem* 251(9): 2842-2847; 1976.

Polyadenylate-rich mRNA from a line of functional rat pituitary tumor cells (GH_3) directed protein synthesis in a cell-free translation system from wheat embryos. The products of translation, when analyzed by electrophoresis and fluorography, did not include any protein comigrating with rat prolactin. One prominent protein synthesized was precipitated using antiprolactin antiserum. This protein was larger than the prolactin standard (apparent molecular wt 28,000 ± 2,000). Addition of a GH_3 cell extract to the translation system resulted in the appearance of a protein that comigrated with rat prolactin and was immunoprecipitated with anti-rat prolactin antiserum. The presence of thyrotropin-releasing factors in spinner cultures of GH_3 cells for 48 to 72 hr increased de novo prolactin biosynthesis. Levels of mRNA coding for prolactin, determined by translation of total mRNA and measurement of preprolactin synthesized, were stimulated 3- to 6-fold relative to the levels in control cultures, suggesting that regulation of prolactin synthesis occurs, at least in part, at a transcriptional level.

3509 LIPID COMPOSITION OF EXPERIMENTAL ASTROCYTOMAS ORIGINATING FROM TRANSFORMED RAT AND HAMSTER ASTROCYTE CULTURES. (Eng.) Hauser, G. (McLean Hosp., Belmont, MA 02178); Eichberg, J.; Shein, H. M. *Brain Res* 109(3): 636-642; 1976.

The lipid patterns of several tumors produced by astroglial cell lines transformed with chemical or viral agents are presented and compared with those of tumors produced when the cells were injected sc into hamsters. Three of the lines were derived from adult rat astrocytes that had been transformed in situ with N-nitrosomethylurea and subsequently cloned. These three lines, C-3, C-6, and $C\text{-}2_1$, contained, respectively, questionable, substantial, and small amounts of S-100 protein. The other two lines originated from newborn (NT 1) or fetal (THA 15) Syrian hamster astrocytes transformed with simian virus 40. Among the rat tumors, the total lipid content ranged from 18.7 to 27.1 mg/g wet wt. The C-6 and $C\text{-}2_1$ tumors contained less total lipid and triglycerol than the least differentiated C-3, and more glycolipid and phospholipid. The patterns of the two hamster tumors were fairly similar to each other except for the triglycerol content, which varied from 4.9% to 14.1% of the total lipid. No significant amounts of cholesterol esters were detected in any of the tumors. The phospholipid patterns were similar in all tumors, regardless of species origin or degree of differentiation. Injection of spontaneously transformed immature hamster astrocytes into hamsters resulted in the growth of tumors with very similar lipid patterns, although the tumors contained a substantially lower lipid:protein ratio. The tumors contained somewhat more triacylglycerol and less glycolipid, but the tumors and cells contained identical proportions of phospholipid and closely comparable phospholipid compositions. (31 references)

3510 SPECIFIC BINDING OF [^{3}H]OESTRADIOL BY CYTOPLASMIC COMPONENTS OF FEMALE RAT LIVER.

(Eng.) Powell-Jones, W. (Welsh Natl. Sch. Medicine, Heath Park, Cardiff, CF4 4XX, Wales); Davies, P.; Griffiths, K. *J Endocrinol* 69(1): 167-168; 1976.

Mammary tumors were induced in virgin female Sprague-Dawley rats, and specific estradiol binding components in the cytoplasmic liver extracts were analyzed. Saturation analysis was performed by incubating aliquots of cytosol with varying concentrations (45-1,000 pmol/l) of 2,4,6,7-^{3}H-estradiol with and without a hundredfold excess of diethylstilbestrol. Sedimentation analysis of the preparations showed two peaks of radioactivity corresponding to labeled steroid-protein complexes of around 4S and 8S. The 8S peak could be displaced by a hundredfold excess of unlabeled estradiol or diethylstilbestrol with a concomitant increase in the 4S peak. No displacement occurred with high concentrations of progesterone, 5α-dihydrotestosterone, or dexamethasone. Liver cytosols from normal and tumor-bearing rats showed similar patterns. Saturation analysis of the binding of estradiol to low capacity components indicated a dissociation constant of the steroid-protein complex in the range of 900-2400 pmol/l and a receptor concentration of 50-100 fmol/gl of protein.

3511 CONTROL OF THE MITOTIC ACTIVITY IN THE RAT SEMINAL VESICLE BY ''CHALONE.''

(Eng.) Soederstrom, K.-O. (Dept. Anatomy, Univ. Turku, 20520 Turku 52, Finland); Tuohimaa, P. *Acta Endocrinol* 81(3): 668-672; 1976.

One month after castration, male Sprague-Dawley rats were injected daily with 2 x 50 μg of testosterone. Starting before the highest mitotic activity of seminal vesicles, three injections of normal seminal vesicle extract were given into the lumen at 3-hr intervals. The contralateral seminal vesicle was injected with rat liver extract. ^{3}H-thymidine was given iv. The labeling index of the seminal vesicles was determined by radioautography. The crude supernatant of seminal vesicle inhibited the mitotic activity of the seminal vesicles most effectively. The mean decrease was 34%. The liver extract did not influence the mitotic activity. When the seminal vesicle extract was fractionated by Sephadex G-25, the largest fraction (molecular wt over 3,000) showed the fraction (molecular wt under 1,000) was ineffective. The inhibition of mitosis could also be induced by an unspecific factor, since pronase showed slight inhibitory action on seminal vesicle mitosis. However, the seminal vesicle extract had no inhibitory action on the epidermal mitotic activity.

3512 SHUTTLES FOR THE TRANSFER OF REDUCING EQUIVALENTS IN EHRLICH ASCITES TUMOR CELLS.

(Eng.) Eboli, M. L. (Inst. General Pathology, Catholic Univ., Via Pineta Sacchetti 644, 00168 Rome, Italy); Galeotti, T.; Dionisi, O.; Longhi, G.; Terranova, T. *Arch Biochem Biophys* 173(2): 747-749; 1976.

The oxidation of lactate to pyruvate was studied in wild strain Ehrlich ascites tumor cells. Aminooxyacetate did not affect the production of pyruvate under conditions of endogenous metabolism, whereas it partially inhibited in the cells supplemented either with α-oxoglutarate plus aspartate or with DL-isocitrate. Reconstitution experiments of the fatty acid cycle performed in isolated mitochondria showed that added NADH was actively oxidized. Whereas the operation of the malate-aspartate shuttle under conditions of endogenous metabolism is ruled out, a possible role of the fatty acid shuttle in the transport of reducing equivalents across the mitochondrial membrane is suggested.

3513 THE AGE-RELATED OCCURRENCE OF WAX ESTERS IN THE MOUSE PREPUTIAL GLAND TUMOUR.

(Eng.) Grigor, M. R. (Biochemistry Res. Group, Univ. Otago, Dunedin, New Zealand). *Biochim Biophys Acta* 431(1): 157-164; 1976.

The changes in lipid composition of the mouse preputial gland tumor (ESR-586) during its growth in vivo in C57BL mice were studied. The tumor accumulated wax esters from between 20 and 25 days after transplantation until they became the most abundant lipid class. Prior to this time, wax esters were not detectable. The process occurred in both male and female host mice and appeared to be determined by a response of the host to the tumor, rather than by a property of the tumor itself. The most abundant fatty alcohol present in the wax esters was hexadecanol. This contrasts with the greater proportions of the C_{20} to C_{24} chains found for the alkyl portion of the alkyldiacylglycerols, for which the precursors are fatty alcohols.

3514 CHARACTERIZATION OF THE ANDROGEN RECEPTOR FROM A SYRIAN HAMSTER DUCTUS DEFERENS TUMOR CELL LINE (DDT_1).

(Eng.) Norris, J. S. (Baylor Coll. Medicine, Houston, TX 77025); Kohler, P. O. *Science* 192(4242): 898-900; 1976.

The complex cytoplasmic androgen receptor protein, which binds ^{3}H-labeled 5,α-dihydrotestosterone, of the hamster ductus deferens cloned tumor cell line (DDT_1) was characterized. Diethylaminoethyl (DEAE)-cellulose chromatography of cytosol from these cells yielded two major receptor peaks of activity. Identification of this steroid binding protein as a cytoplasmic receptor was confirmed by salt dissociation on sucrose gradients, stability of the hormone-receptor complex at 0 C, and the retention patterns on phosphocellulose and DEAE cellulose. It is proposed that the cytoplasmic form of the receptor is a 6S dimer, consisting of an A and B monomer, which moves into the nucleus and is bound to the chromatin acceptor site by the B subunit. The A subunit then dissociates from the B monomer and searches along adjacent chromatin regions until it locates and binds to a specific regulatory site on naked DNA. This model would explain the presence of subunits with different functions, but each capable of binding steroid hormone.

3515 UPTAKE OF MELANIN PRECURSORS IN MOUSE MELANOMA. (Eng.) Hutton, I. M. (Battle Hosp., Reading, Berks, England); Pentland, M. *Clin Oncol* 2(1): 17-23; 1976.

The uptake of tritium-labeled precursors of melanin by mouse malignant melanoma was measured. Harding-Passey melanoma was implanted into 59 6-wk-old female CBA/CA mice and allowed to grow for 7-10 days. Tritiated dihydroxyphenylalanine (Dopa, 25 Ci) was injected sc, and the animals were killed 2, 4, 6, or 16 hr after injection. The uptake of tritiated Dopa by tumor, kidney, liver, skin, spleen, and lung was determined by liquid scintillation, and tissue uptakes were calculated as a percentage of the uptake in muscle. Dopa was taken up in higher concentration by the tumor than by any other tissue except kidney. Dihydroxyphenylethylamine (Dopamine), a precursor of the catecholamines, was poorly concentrated despite close chemical similarity to Dopa. This may indicate specificity of uptake of direct melanin precursors. Such a technique may be developed in the future for tumor scanning and cytotoxic therapy.

3516 LOW TEMPERATURE SPECIFIC HEAT ANOMALIES IN MELANINS AND TUMOUR MELANOSOMES. (Eng.) Mizutani, U. (Carnegie-Mellon Univ., Mellon Inst., 4400 Fifth Ave., Pittsburgh, PA 15213); Massalski, T. B.; McGinness, J. E.; Corry, P. M. *Nature* 259(5543): 505-507; 1976.

Specific heat measurements were carried out on intact melanosomes isolated from human malignant melanoma cells, melanin, and synthetic melanin. Synthetic melanins were prepared by adding 20% diethylamine to a starting mixture of L-dopa and twice-distilled water. The values of the β coefficient (calculated from $C = \mathrm{gamma}T + \mathrm{beta}T^3 + \mathrm{delta}T^5$) indicated that the three materials are quite similar in their lattice contribution to the specific heat. The smallest linear term gamma occurred in the synthetic melanin, and the highest value occurred in the biological extract from the human tumor. The close agreement in the phonon contributions for the different melanins suggests that the change in the linear term coefficient is unlikely to be due to a change in the phonon spectrum. A discontinuity in the heat capacity at about 1.9 K for all melanins measured was thought to be associated with a magnetic phase transition. These effects occur in synthetic as well as natural melanins, but are most pronounced in the melanosomes isolated from the human melanoma.

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National Cancer Institute

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PREFACE

Carcinogenesis Abstracts is a publication of the National Cancer Institute. The journal serves as a vehicle through which current documentation of carcinogenesis research highlights are compiled, condensed, and disseminated on a regular basis. It represents an integral part of the Institute's program of fostering and supporting coordinated research into cancer etiology. Issues of *Carcinogenesis Abstracts* normally contain three-hundred abstracts and three-hundred citations (unaccompanied by corresponding abstracts). Abstracts and citations refer to the current scientific literature that describes the most significant carcinogenesis research carried on at the National Cancer Institute, other governmental agencies, and private institutions. *Carcinogenesis Abstracts* is intended to be a highly useful current awareness tool for scientists engaged in carcinogenesis research or related areas. The great number and diversity of publications relevant to carcinogenesis make imperative the availability of this service to investigators whose work requires that they keep abreast with current developments in the field.

Carcinogenesis Abstracts is normally published monthly. Volume XIV covers the scientific literature published from Jan 1976 through Dec 1976. To increase the usefulness of *Carcinogenesis Abstracts*, Volume XIV, a Wiswesser Line Notation index and a Chemical Abstracts Service Registry Number index have been provided. These indexes reference compounds described in abstracted articles. A cumulative subject, author, CAS Registry Number, and Wiswesser Line Notation index for Volume XIV will be published shortly after the final regular issue.

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NOTE

Journal names are abbreviated according to the list of abbreviations used by *Index Medicus*. For journals not covered by *Index Medicus*, the abbreviations found in *Chemical Abstracts Service Source Index*, 1907-1974 Cumulative, are used. New journals are verified in *New Serial Titles* and abbreviated according to *International Standard ISO 833*.

LANGUAGE ABBREVIATIONS

Afr.	Afrikaans
Ara.	Arabic
Bul.	Bulgarian
Chi.	Chinese
Cro.	Croation
Cze.	Czech
Dan.	Danish
Dut.	Dutch
Eng.	English
Est.	Estonian
Fin.	Finnish
Fle.	Flemish
Fre.	French
Geo.	Georgian
Ger.	German
Gre.	Greek
Heb.	Hebrew
Hun.	Hungarian
Ice.	Icelandic
Ind.	Indonesian
Ita.	Italian
Jpn.	Japanese
Kor.	Korean
Lav.	Latvian
Lit.	Lithuanian
Nor.	Norwegian
Pol.	Polish
Por.	Portuguese
Rum.	Rumanian
Rus.	Russian
Ser.	Serbo-Croatian
Slo.	Slovak
Spa.	Spanish
Swe.	Swedish
Tha.	Thai
Tur.	Turkish
Ukr.	Ukrainian
Vie.	Vietnamese

ABBREVIATIONS USED IN ABSTRACTS

A	angstrom(s)
ACTH	adrenocorticotropic hormone
ADP	adenosine diphosphate
AMP	adenosine monophosphate
ATP	adenosine triphosphate
approx	approximately
av	average
BCG	bacillus Calmette-Guerin
bid	twice daily
C	degree(s) centigrade
cal	calorie(s)
kcal	kilocalorie(s)
cc	cubic centimeter(s)
Ci	curie(s)
mCi	millicurie(s)
μCi	microcurie(s)
cm	centimeter(s)
CNS	central nervous system
cpm	counts per minute
DNA	deoxyribonucleic acid
ED_{50}	median effective dose
EDTA	ethylenediamine tetraacetic acid
g	gram(s)
kg	kilogram(s)
mg	milligram(s)
μg	microgram(s)
Hb	hemoglobin
hr	hour(s)
ia	intra-arterial
id	intradermal
ILS	increased life span
im	intramuscular
ip	intraperitoneal
IU	International Unit(s)
iv	intravenous
Km	Michaelis constant
LD	lethal dose
LD_{50}	median lethal dose
M	molar
μM	micromolar
mOsm	milliosmolar
max	maximum
mEq	milliequivalent(s)
min	minute(s)
ml	milliliter
μl	microliter(s)
mm	millimeter(s)
mo	month(s)
N	normal concentration
NAD	nicotinamide adenine dinucleotide
NADH	reduced nicotinamide adenine dinucleotide
NADP	nicotinamide adenine dinucleotidephosphate
NADPH	reduced nicotinamide adenine dinucleotide-phosphate
NCI	National Cancer Institute
NIH	National Institutes of Health
PAS	periodic acid--Schiff
po	orally
ppb	parts per billion
ppm	parts per million
R	roentgen
RBC	red blood cells (erythrocytes)
RNA	ribonucleic acid
rpm	revolutions per minute
sc	subcutaneous
sec	second(s)
SGOT	serum glutamic-oxalacetic transaminase
SGPT	serum glutamic-pyruvic transaminase
soln	solution
TCD	tissue culture dose
TCD_{50}	median tissue culture dose
tid	three times daily
UV	ultraviolet
vol	volume
WBC	white blood cells (leukocytes)
wk	week(s)
wt	weight
x	times
yr	year(s)

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3601 A SIMPLE METHOD FOR DETECTING ENVIRONMENTAL CARCINOGENS AS MUTAGENS.

(Eng.) McCann, J. (Dept. Biochemistry, Univ. California, Berkeley, CA 94720); Ames, B. N. *Ann NY Acad Sci* 271: 5-13; 1976.

A large amount of evidence that carcinogens are mutagens has been obtained by a sensitive and simple bacterial test: of 139 carcinogens tested, 117 were detected as mutagens; of 59 noncarcinogens, only 5 were mutagenic. The test uses various strains of specially constructed mutants of *Salmonella typhimurium* that are reverted to prototrophy by base-pair substitutions (in strains TA1535 and TA100) or by frame shifts (in strains TA1538, and TA98). TA98 and TA100 detect substances that cause nicks in DNA, and mutagenesis occurs as a consequence of an error-prone repair system enhanced by the introduction of a resistance transfer factor. By adding a microsomal activation system of rat (or human) liver to the petri dishes in which the mutagenicity is assayed, a variety of carcinogens can be activated to mutagens in a manner similar to that in vivo. The addition of β-glucuronidase to the petri dishes allows the detection of glucuronidated mutagens in urine samples. Use of the assay indicated that the food additive, furylfuramide, and the gasoline additive, ethylene dibromide, had mutagenic properties, although neither agent was believed to be carcinogenic. Thorough testing has since demonstrated that these materials are indeed carcinogens. The use of short-term, efficient test systems such as the *Salmonella*/microsome system can help to pinpoint, among the thousands of chemicals to which humans are exposed, those that should be given priority in more extensive, long-term animal tests. (29 references)

3602 LABORATORY APPROACHES TO THE IDENTIFICATION OF CARCINOGENS.

(Eng.) Bates, R. R. (Natl. Cancer Inst., Frederick Res. Center, Frederick, MD 21701). *Ann NY Acad Sci* 271: 29-38; 1976.

Possible approaches for detecting carcinogens are discussed. Animal trials remain the most definitive test, but the expense can be prohibitive when large numbers of materials are to be tested. In vitro approaches include: the identification of direct alkylating agents by the reagent 4-nitrobenzoylpyridine; assay of the ability of substances to interact covalently with DNA; determination of the ability to elicit DNA repair synthesis; determination of the mutagenicity of substances, particularly in microorganisms, *Drosophila*, and cultured cells; and assay of the ability to transform cultured mammalian cells. These tests can only be employed as tools for deciding which chemicals should have the highest priority for in vivo testing. (67 references)

3603 PREDICTIVE VALUE OF MUTAGENICITY TESTS IN CHEMICAL CARCINOGENESIS.

(Eng.) Bartsch, H. (International Agency Res. Cancer, 150 Cours Albert Thomas, 69008 Lyon, France). *Mutat Res* 38(3): 177-189; 1976.

The value of mutagenic assays in predicting carcinogenic properties is discussed. Electrophilicity is a common property of the ultimate reactive metabolites of known carcinogenic chemicals. Results with a rat liver microsome test in vitro with *Salmonella typhimurium* strains have shown that approximately 80%-90% of the carcinogens tested were also mutagens, with false positives and false negatives occurring in 10%-15% of the results. The application of these mutagenic tests to vinyl chloride and related agents and to the nitrosamines is discussed. The identification of people who may be at exceptionally high risk for cancer because of a genetically determined enzyme profile may in the future be of some assistance in establishing regulatory mechanisms to limit environmental carcinogen levels. Despite the tendency of chemicals to be both carcinogens and mutagens, it is not known whether in the future all carcinogens will be found to be mutagenic and all mutagens carcinogenic. Up to now, no carcinogenic effects of the strong mutagens such as base analogs, nitrous acid, and hydroxylamine have been reported. Conversely, synthetic steroidal sex hormones, carcinogenic in animals, have not yet been reported to be mutagenic. (68 references)

3604 HISTORICAL EVOLUTION OF SHORT-TERM TESTS FOR CARCINOGENICITY.

(Eng.) Clayson, D. B. *In: Carcinogenesis Testing of Chemicals. Proceedings, Conference on Carcinogenesis Testing in the Development of New Drugs, May 23-25, 1973, Washington, D.C.* (Cleveland: CRC Press, Inc.): pp. 79-89; 1973.

Problems encountered in the development of short-term carcinogenicity tests are reviewed. In test development, the limits within which a test is operative should be defined carefully; the test should incorporate some specific attribute of the carcinogen or of the carcinogenic process; and known carcinogens should be positive and known noncarcinogens negative within test limits. The specificity of the sebaceous gland suppression, hyperplasia, and formazan deposition skin tests is in doubt. Although the bladder hyperplasia tests have been successful, the different biological processes that lead to the hyperplasia do not seem to encourage a wider application for this test. Liver tests to screen nitrosamines require excessive amounts of the test substance, which might induce other tumors in other locations. Tests in which enhanced tumor incidence is assayed against a relatively high natural tumor incidence are difficult to interpret; hypothetical age-versus-incidence curves show that the time of test termination greatly influences the apparent significance of the results. In vivo short-term tests seem of little use for screening large numbers of unknown drugs with unknown activity and tissue specificity. In vitro short-term tests should mimic part or all of the carcinogenic process in the living animal. Although host-mediated assay systems are limited by the tissue from which the microsomes are obtained and by the cofactors chosen by the investigator, this type of test seems likely to be a future research tool. Difficulties with in vitro carcinogenesis studies include: possible inability of the cell to activate the carcinogen, interference by possible oncogenic viruses in the animal, and the fact that the techniques are time-consuming and complicated. The use of macromolecular carcinogen binding and DNA repair studies depends on recognizing the kind of DNA damage induced by various types of carcinogens. Tumor progression tests appear unsuccessful. An acceptable short-term screening test will probably not be developed for many years. (59 references)

3605 OVERVIEW: ENVIRONMENTAL FACTORS. (Eng.) MacMahon, B. *In: Persons at High Risk of Cancer. An Approach to Cancer Etiology and Control.* Edited by J. F. Fraumeni, Jr. (New York: Academic Press, Inc.): pp. 285-290; 1975.

An important feature emerging from this review is the large number and variety of known carcinogens: their nature (chemical, physical, or biological), their modes of action, and the age at which they exert their effects. With respect to prevention, no single known measure would lengthen the life or improve the health of the American population more than elimination of cigarette smoking, which would reduce current lung cancer rates by about one-tenth. There is also a need to reduce radiation exposure and exposure to known carcinogens in industry. The nature and variety of recognized environmental factors suggest some of the difficulties facing research efforts to uncover causes. Difficulties arise from the ubiquity of some possible determinants and the problems of identifying and measuring factors that are often seemingly trivial and present in minute amounts. The time scale involved in carcinogenesis (the latent period may be quite short for some cancers and measured in decades for others) also presents a problem for research. Because of the complexity of the situation, one must encourage trained investigators with imagination and initiative to explore new fields. (12 references)

3606 CHEMICAL CARCINOGENS. (Ger.) Rademacher, P. (Organisch-Chemisches Institut, Universitaet Muenster, Muenster, West Germany). *Chemie in unserer Zeit* 9(3): 79-84; 1975.

The roster of known chemical carcinogens, demonstrations of their properties, and their mechanistic development in host cells are reviewed with special attention to occupational exposure. If 90% of all cancers result exogenously, then 60% are attributed to chemical hazards; cancer mortality is 25% higher among chemists than the general population. The conventional biphasic schedule of cell transformation is adopted. During an initial phase of chemical attack, a normal cell becomes precancerous; during a subsequent promotional phase, cocarcinogens shift the atypical cell into the malignant state. Experimental evidence fitting various chemical carcinogens into this scheme is cited. Inorganic compounds, aromatic hydrocarbons and their N, O, and S analogs, aromatic amines, nitro and azo compounds, aliphatic hydrazines, azo and azoxy compounds, N-nitroso compounds, alkylating agents, naturally occurring products (especially bacterial, fungal, or faunal metabolic products), and routine laboratory reagents are implicated. Based on the recent vinyl chloride episode, maximum allowable occupational concentrations are discussed in connection with instrumentation and procedures designed to minimize vulnerability to carcinogenic hazards. (14 references)

3607 CHEMICAL CARCINOGENS IN INDUSTRY. (Eng.) Carter, R. L. (Inst. Cancer Res., Royal Cancer Hosp., London, England); Roe, F. J. C. *J Soc Occup Med* 25(3): 86-94; 1975.

Occupational hygiene programs and industrial plant designs must be improved to reduce the hazards of known industrial carcinogens. Polycyclic aromatic hydrocarbons, specifically, mineral oils and coal gas, have been linked with scrotal and skin cancers in industrial workers. A more refined cutting oil may reduce the risk of scrotal cancer. The incidence of deaths from lung cancer in England was found to be almost double the normal rate for workers exposed to coal and tar gas. An enhanced risk for bladder cancer was noted in workers exposed to benzidine, β-naphthylamine, and α-naphthylamine. The incidence was dose-related, with the highest rate in workers who distilled β-naphthylamine. Exposure to the alkylating agent mustard gas was associated with a marked increase in deaths from respiratory tract carcinoma in Japanese workers. Bis(chloromethyl)ether (BCME) exposure for 6 to 9 yr in 68 production workers (31-65 yr) was followed by lung cancer in 8/68; 5 tumors were oat-cell carcinoma. An increased incidence of hepatic angiosarcoma was noted for workers involved in polymerizing vinyl chloride to polyvinyl chloride (PVC); there was also an excess of neoplasms of the biliary system, lung, and brain in vinyl chloride workers who died before age 60. An increased risk for respiratory cancer has been established in workers involved in mining or milling asbestos products; the basis for carcinogenicity is still uncertain, but studies indicate that there may be an optimal size of the fiber for carcinogenic activity. Computerized industrial epidemiology may help to determine cancers of occupational origin that have been overlooked and help to protect workers from known hazards. (72 references)

3608 OCCUPATION. (Eng.) Cole, P.; Goldman, M. B. *In: Persons at High Risk of Cancer. An Approach to Cancer Etiology and Control.* Edited by J. F. Fraumeni, Jr. (New York: Academic Press, Inc.): pp. 167-184; 1975.

Epidemiological studies of occupational carcinogenesis are reviewed, carcinogenic agents in the occupational environment are tabulated by agent and site of cancer, and the control of occupational carcinogenesis is briefly discussed. Problems involved in occupational studies include the long incubation period (20 yr or more), workers are unaware of conditions surrounding them or the specific substances handled, and the close association between occupation and social class, which is itself a correlate of risk for many cancers. The magnitude of the excess risk and temporal aspects of chemical carcinogenesis has been worked out best for organic agents, especially the aromatic hydrocarbons. Several heavy metals, fibers, and dusts have also been implicated in occupational carcinogenesis, but not as persuasively as the aromatic hydrocarbons. Existing epidemiologic methods are adequate for demonstrating carcinogenic hazards in the work environment even when the excess risk is rather small and the disease in question is relatively common in the general population. This capacity for recognition has, by itself, no preventive or control value. Technical progress to prevent occupational cancer is now advancing rapidly. More fundamental, however, is the plan to use lower organisms or laboratory animals to screen compounds of potential industrial value before they arrive in the workplace. With respect to mortality reduction, both technical and epidemiologic methods seem likely to be useful, the latter to be in the form of improving the design and evaluation of proposed early disease detection programs.

3609 DOSE-RESPONSE RELATIONSHIP AND THRESHOLD CONCEPTS. (Eng.) Kotin, P. (Health, Safety & Environment Dept., Johns-Manville Corp., Denver, CO 80217). *Ann NY Acad Sci* 271: 22-28; 1976.

The concepts of dose-response and threshold levels in cancer induction are examined. The applicability of the dose-response concept has been clearly demonstrated for the tumorigenic effects of carcinogenic agents in animal models over certain concentration ranges. This has been verified in man, and dose-response data and no-effect levels have been determined for carcinogenic organic and inorganic chemicals (specifically for vinyl chloride and asbestos), metals, and for non-ionizing and ionizing radiation. In the absence of evidence to the contrary, the applicability of the threshold concept in considerations of acceptable levels of carcinogens within the environment cannot be denied. (23 references)

3610 HEALTH EFFECTS OF FOSSIL FUEL COMBUSTION PRODUCTS: REPORT OF A WORKSHOP. (Eng.) Comar, C. L. (Electric Power Res. Inst., P.O. Box 10412, Palo Alto, CA 94303); Nelson, N. *Environ Health Perspect* 12: 149-170; 1975.

The biological impact of fossil fuel combustion and air pollution must be determined regarding their carcinogenic and mutagenic potential. Carcinogenic agents identified in fossil fuel combustion products include polycyclic and other aromatic hydrocarbons, trace metals, radionuclides, and particulates. Factors that would support the argument that air pollution is a causal factor in lung cancer are: polluted air contains known carcinogens; lung cancer in urban areas and in some international migrant groups can be associated with urban residence; workmen occupationally exposed to benzo(a)pyrene, asbestos, and other pollutants have excess cancer; and cigarette smoking, which has ingredients similar to urban pollution, causes lung cancer. Factors that would argue against urban air pollution as a cause of cancer are: the differential excess due to the urban factor has not been detected as an absolute excess in those areas with the greatest urban pollution; lung cancer rates in lifetime urban residents are not higher than in migrants to urban areas; air pollution does not appear to affect women as much as men; and lung cancer rates do not correlate with urban pollution by benzo(a)pyrene. The most urgent goals for air pollution control are in control of soot, suspended particular matter, and products of incomplete combustion. Priority should be given to the study of persons occupationally exposed to suspected carcinogens and to the study of other population groups who are exposed to point sources and whose health can be monitored regularly. (47 references)

3611 THE CHLOROETHERS--OCCUPATIONAL CARCINOGENS: A SUMMARY OF LABORATORY AND EPIDEMIOLOGICAL STUDIES. (Eng.) Nelson, N. (Inst. Environmental Medicine, New York Univ., New York, NY 10016). *Ann NY Acad Sci* 271: 81-90; 1976.

A brief review is presented of structure-activity relationships, usage in technology, environmental occurrence, and carcinogenic activity of the chloroethers. Bis(chloromethyl) ether (BCME) and chloromethyl methyl ether (CMME) have been shown to be carcinogenic in rodents; for example, when female mice (Sprague-Dawley and ICR/Ha Swiss) were treated topically with 1 mg of chloroether followed by 25 μg of promotor phorbol myristate (three times per week), five papillomas and one squamous carcinoma developed with CMME and five papillomas and two squamous carcinomas developed after BCME. The chloroethers were shown to be direct-acting mutagens in several bacterial systems. In a study involving 1,800 persons occupationally exposed to CMME, the age adjusted death rate for lung cancer was found to be 1.48/1,000/yr, significantly greater than the rate of 0.59/1,000/yr determined for a control group. (23 references)

3612 MULTIPLE FACTORS IN CARCINOGENESIS. (Eng.) Bingham, E. (Dept. Environmental Health, Kettering Lab., Univ. Cincinnati Medical Center, Cincinnati, OH 45267); Niemeier, R. W.; Reid, J. B. *Ann NY Acad Sci* 271: 14-21; 1976.

The interaction of multiple factors in carcinogenesis is discussed. Exposure of an individual to a carcinogenic agent can be modified by genetic determinants, such as enzyme levels, immune defense capacity, and hormonal imbalance; cultural influences, such as smoking, diet, and other environmental factors; and health status. As examples of such modification, smoking synergistically increases lung cancer incidence in asbestos workers and uranium miners, and high dietary levels of riboflavin reduce the carcinogenic activity of 4-dimethylaminoazobenzene in rats. Exposure to more than one carcinogenic agent also influences cancer incidence: for example, the oral administration of benzo(a)pyrene and a nitrosamine results in a higher incidence of lung and stomach tumors (in an unidentified species) than if each agent is administered separately. The magnitude of a cocarcinogenic effect may not always be appreciated; a 1,000-fold enhancement of the effective concentration of benzo(a)pyrene for skin tumor induction is demonstrated by use of a cocarcinogenic solvent such as n-dodecane. Because of the complex interaction of multiple factors that govern the environmental risk of cancer in man, causal relationships and correlations may be difficult to define conclusively. Negative results do not necessarily mean that an agent is inactive, and no single protocol is valid for testing all suspected carcinogens. Also, multiple factors may control the latent period, and the risk assessment of low levels of weak carcinogens is very difficult. Therefore, at present the setting of standards for carcinogens must be based on no exposure. (58 references)

3613 AIR POLLUTION. (Eng.) Pike, M. C.; Gordon, R. J.; Henderson, B. E.; Menck, H. R.; SooHoo, J. *In: Persons at High Risk of Cancer. An Approach to Cancer Etiology and Control.* Edited by J. F. Fraumeni, Jr. (New York: Academic Press, Inc.): pp. 225-239; 1975.

The role of air pollution in carcinogenesis, particularly lung cancer, is reviewed. Measurements of benzo(*a*)pyrene (BP) concentrations in the atmosphere provide the most widely available indicator of polycyclic aromatic hydrocarbons, the main class of chemically identified carcinogens in the air. Two interferences in evaluating the possible effect of air pollution on lung cancer are cigarette smoke and occupational exposure to lung carcinogens. The results of a study of carbonization workers in a British gasworks indicate that each nanogram per cubic meter of BP causes $0.4/10^5$ extra lung cancer cases per year, so that a city with 50 ng/m^3 might expect to have an extra $18/10^5$ lung cancer cases per year. This estimate of a small general air pollution effect on lung cancer agrees with most other epidemiological evidence. Another study compared the lung cancer rates in Liverpool to those in rural North Wales and found that the urban effect produced an excess of $28/10^5$ lung cancer deaths in nonsmokers and $100/10^5$ such deaths in smokers. The difference in BP levels in the two areas was 70 ng/m^3, leading to the estimate that the air pollution effect in the presence of cigarette smoking was $1.4/10^5$ per ng/m^3 BP or $0.4/10^5$ per ng/m^3 BP in nonsmokers. These two studies support the notion of a proportional relationship between BP concentration in the air and lung cancer rate. Bladder and scrotal cancer are also linked to high levels of air pollution. Calculations are included that show that the excess lung cancer rate per ng/m^3 BP in air could be as much as 3.5 times greater in smokers than in nonsmokers; the differential effect apparently is not related to the absolute level of smoking. The intense, short exposure to polycyclic aromatic hydrocarbons through smoking a cigarette may maximally induce the aryl hydrocarbon hydroxylase (AHH) system in the lung. The low-level carcinogens in the air are then optimally transformed to their active form and do their greatest damage. The nonsmoker has a lower level of AHH activity in the lung, and thus air pollution carcinogens might be cleared via a less carcinogenic route. (38 references)

3614 TOBACCO. (Eng.) Hammond, E. C. *In: Persons at High Risk of Cancer. An Approach to Cancer Etiology and Control.* Edited by J. F. Fraumeni, Jr. (New York: Academic Press, Inc.): pp. 131-138; 1975.

Selected aspects of tobacco-related cancer are reviewed and areas for future study suggested. A prospective epidemiological study of over one million men and women between October 1959 and September 1965 gave findings consistent with dozens of other studies; that is, cigarette smoking increases the risk of lung cancer to a greater degree than any other cancer site, multiplies the risk of cancer of the buccal cavity and pharynx by a factor of about 3-10, and increases the risk of cancers of the larynx, esophagus, bladder, and pancreas. Heavy alcohol consumption increases the risk of mouth and throat cancer even further in smokers. Lung cancer death rates increase with the amount of cigarette smoking and with depth of inhalation and are far higher among people who started smoking in their early teens. Men with a lifetime history of only pipe or cigar smoking have a significantly higher lung cancer death rate than men who never smoked, but a considerably lower rate than men who smoked even a few cigarettes a day. This is explained by the fact that most pipe and cigar smokers do not inhale, while most cigarette smokers do. Lung cancer death rates are lower among ex-cigarette smokers than those who continue the habit and are lower among those who have not smoked for many years than those who have not smoked for a few years. Further research should be aimed at the following two questions: (a) does reduction in the tar and/or nicotine content of cigarettes significantly reduce the risk of cancer; and (b) is there an accompanying decrease or increase in the risk of death from other serious diseases, especially coronary heart disease. Preliminary evidence suggests that tar and nicotine in cigarette smoke are mainly responsible for the cancer producing effects and that reduced exposure can lower the risk of cancer among people who were heavily exposed for many years. Death rates from coronary heart disease, however, were a little higher in men who smoked low tar and nicotine cigarettes than in men who smoked high tar and nicotine cigarettes. This finding may be due to biasing factors that need to be evaluated. (14 references)

3615 CANCER OF THE LUNG. (Eng.) Cantor, R. I.; Weiss, A. J. *In: Oncologic Medicine: Clinical Topics and Practical Management.* Sutnick, A. I.; Engstrom, P. F., eds. (Baltimore: University Park Press): pp.109-123; 1976.

Discussion is presented on the etiology, classification, clinical manifestations, diagnostic procedures, tumor markers, and treatment of lung cancer. Etiological factors may act directly, at the epithelial level, or indirectly, by diminishing normal pulmonary cleansing. The role of cigarette smoking seems indisputable, the hazard of tumor occurrence increasing proportionately with the intensity and duration of exposure. Termination of exposure diminishes the hazard, with reversal of epithelial atypia in more than 90% of cases. Unfortunately, this is not the case for occupational induction of lung cancer, in which termination of exposure does not seem to reduce appreciably the later incidence of cancer, although it does prevent a further increase in hazard. Consideration is given to the use of cytology, bronchoscopy, and mediastinoscopy as diagnostic adjuvants and the the association of carcinoembryonic antigen (in 75% of cases) and α-fetoprotein (in 7% of cases) with bronchogenic cancer. The following therapeutic approaches are discussed: radiation therapy, surgery, chemotherapy (single agent and combination), and immunotherapy. Before a decision is made to pursue a curative procedure, the patient must be evaluated with respect to physiologic qualifications and the presence of biologic factors that indicate a poor outcome: (a) undifferentiated small cell carcinoma, which is a systemic disease; (b) hilar or mediastinal adenopathy; (c) direct mediastinal extension; (d) pleural effusion, especially if malignant cells can be identified; and (e) metastatic disease to any area beyond the primarily involved hemithorax. (33 references)

3616 CANCER OF THE LUNG: HOST AND ENVIRONMENTAL INTERACTION. (Eng.) Tokuhata, G. K. *In: Cancer Genetics*. Edited by H. T. Lynch. (Springfield, IL: Charles C. Thomas): pp. 213-232; 1976.

In addition to smoking and other environmental factors, familial studies have revealed that some inherited genetic factor plays an important role in the etiology of lung cancer. The incidence of smokers among the close relatives of lung cancer victims was found to be greater than that among the relatives of control subjects, and it is suggested that this results from some genetic influence, smokers and nonsmokers being genotypically distinguishable. There is a strong synergistic interaction between smoking and an inherent susceptibility to lung cancer (the familial host factor). This is supported by findings that those smokers who have the familial host factor have a 14-fold greater risk of lung cancer than nonsmokers without the factor; smokers without the familial host factor have a 5-fold greater risk, and nonsmokers with the familial factor a 4-fold greater risk, than nonsmokers without the familial host factor. (73 references)

3617 CANCER. U.S. Public Health Service *In: Health Consequences of Smoking 1975.* (Washington: U.S. Dept. of Health, Education, and Welfare): pp. 41-56; 1975.

Clinical, experimental, and epidemiological data collected over the past 30 yr show that the risk of developing lung cancer increases directly with the number of cigarettes smoked per day, total lifetime number of cigarettes smoked, and age at start of smoking. Lung cancer death rates for women have increased dramatically over the past 15 yr coincident with the increasing number of women smokers. Cigarette smoking may interact with occupational exposures, such as uranium mining and asbestos production, to produce a greater risk for lung cancer than with smoking alone. Adenocarcinoma is the most common type of lung cancer in nonsmokers, and squamous cell carcinoma is the most common type when smokers are included. Attempts are being made to identify and remove the carcinogenic factors in cigarettes, which would reduce the risk of developing lung cancer. Chronic respiratory infection may enhance the neoplastic response of the lungs to a systemic carcinogen. All levels of smoking are related to cancer of the bladder, since the bladder stores urine for some time and small amounts of carcinogens in the urine may be sufficient to affect the bladder epithelium. Aryl hydrocarbon hydroxylase (AHH) may be a genetically determined enzyme that mediates increased susceptibility to certain environmental carcinogens; persons with high AHH levels may have an increased risk of lung cancer if they smoke, but no excess risk if they do not smoke. (26 references)

3618 ALCOHOL. (Eng.) Rothman, K. J. *In: Persons at High Risk of Cancer. An Approach to Cancer Etiology and Control.* Edited by J. F. Fraumeni, Jr. (New York: Academic Press, Inc.): pp. 139-150; 1975.

Cancer of specific sites and an increased overall cancer risk attributable to alcohol consumption are reviewed and the prospects for control discussed. Alcohol and tobacco are estimated to account for approximately 3/4 cancers of the oral cavity in U.S. males. The risk of combined heavy smoking and drinking is 15 times as great as that for non-smokers/drinkers, whereas the risk from heavy smoking alone is only 2 to 3 times as great. Alcohol consumption, particularly heavy whiskey consumption, elevates the risk for laryngeal cancer more than 10 times compared to abstinence or moderate drinking. In a study restricted to moderate smokers, it was found that heavy drinkers experienced a risk of esophageal cancer more than 25 times that of nondrinkers. Because the geographic variation of esophageal cancer is exceptional and alcohol consumption is nearly ubiquitous, other factors probably have an important function. The carcinogenic agent elevating the risk in alcohol drinkers may not be alcohol but a contaminant. In cohort studies of alcoholics no excess of stomach cancers was observed, but the distinctive clinical and epidemiologic features of cancer of the gastric cardia, in addition to its reported association with alcohol consumption, indicate that at least some cases may be linked to alcohol consumption. Primary cancer of the liver occurs with great frequency (10%-30%) among alcoholics with cirrhosis. A correlation analysis of age-adjusted mortality data and beer consumption revealed a high correlation between death from rectal cancer and beer consumption, but this association is considered tentative until it can be established in studies with individuals. Heavy drinkers have an estimated 20%-30% increase in cancer risk over nondrinkers and moderate drinkers. To the extent that alcoholics can be approached as a group, it seems advisable to implement trial screening programs to evaluate the potential benefit of early cancer detection and treatment. (36 references)

3619 DRUGS. (Eng.) Hoover, R.; Fraumeni, J. F., Jr. *In: Persons at High Risk of Cancer. An Approach to Cancer Etiology and Control.* Edited by J. F. Fraumeni, Jr. (New York: Academic Press, Inc.): pp. 185-199; 1975.

Epidemiologic evidence for drug-induced cancer in man is reviewed. Radioisotopes exert carcinogenic effects by releasing ionizing radiation at deposition sites in the body. Immunosuppressive agents (antimetabolites, corticosteroids, and antilymphocyte serum) may contribute to the high cancer risk experienced by renal transplant recipients. Cytotoxic agents in cancer chemotherapy are often carcinogenic in laboratory animals and may induce cancer in man. Chlornaphazine was withdrawn from use when high doses for treating polycythemia vera and Hodgkin's disease caused bladder tumors. Certain alkylating agents, such as melphalan or cyclophosphamide, seem to elevate the risk of acute leukemia. A follow-up study of Hodgkin's disease showed that intensive chemotherapy enhanced the risk of second cancers originating at heavily irradiated sites. Prenatal exposure to synthetic estrogens, particularly diethylstilbestrol, has been linked to clear cell carcinomas of the vagina and cervix in over 100 patients (7-29 yr old). Androgenic-anabolic steroids have been implicated in the development of hepatocellular carcinoma after long-term treatment of aplastic anemia. Inorganic arsenicals when taken internally are a cause of skin cancer in man. Other drugs associated with an increased cancer risk in man are phenacetin, coal tar and creosote preparations, diphenylhydantoin, chloramphenicol, amphetamine, and reserpine. A few drugs suggested for epidemiologic study are cancer chemotherapy agents that show carcinogenic potential; tertiary amines, including a number of widely prescribed drugs such as oxytetracycline and chlorpromazine; and iron dextran. (67 references)

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3620 DIET. (Eng.) Berg, J. W. *In: Persons at High Risk of Cancer. An Approach to Cancer Etiology and Control.* Edited by J. F. Fraumeni, Jr. (New York: Academic Press, Inc.): pp. 201-224; 1975.

Carcinogens in the diet, including naturally occurring constituents and food additives, are reviewed and recommendations for gathering further evidence linking diet with specific cancers are suggested. Aflatoxins have been linked with liver cancer in man and are the most potent carcinogens for rats. The major human food item at risk of containing aflatoxins in the U.S. is peanuts. Nitrosamines, which are also powerful carcinogens for animals, are frequently found in foods. A strong epidemiological correlation comes from Japan, where salted fish is an undoubted risk factor for stomach cancer and is also a major source of nitrosamines. Nitrosamines have been reported in low levels in the diets of Africans and Iranians in epidemic esophageal cancer areas. The strongest correlation between polycyclic aromatic hydrocarbons and human cancer is that all populations consuming a great deal of smoked fish seem to be at high risk for stomach cancer. In certain parts of the world, a cancer risk may be posed by natural plant foods. It has been suggested that the high incidence of esophageal cancer in Curacao resulted from the heavy consumption of medicinal teas; carcinogenic activity has now been shown for several tannin-containing extracts of these plants. Other folk medicines contain pyrrolizidine alkaloids that are strong carcinogens. Food additives under study as possible carcinogens include some dyes used to color foods and artificial sweeteners. Food contaminants, including chemical fertilizers and pesticides in vegetable products, antibiotics and growth stimulants in animal products, and substances added during processing and packaging, may all have some carcinogenic risk. The most significant compounds are those that accumulate in the human body, such as asbestos, DDT, and related compounds. Heavy metals are also important carcinogens and may enter the diet in foods and drinking water. Trace elements may affect cancer risk by contribution to a dietary imbalance that favors the development of certain cancers. Certain vitamin deficiencies can enhance or inhibit carcinogenesis in animals. Future studies should concentrate on metabolic epidemiology in animals and man. (156 references)

3621 ARECA NUT. A REVIEW. (Eng.) Arjungi, K. N. (Institut fuer Toxikologie und Chemotherapie am Deutschen Krebsforschungszentrum, Im Neuenheimer Feld 280, 6900 Heidelberg, West Germany). *Arzneim Forsch* 26(5): 951-956; 1976.

Areca catechu Linn, the areca nut, is widely cultivated in Eastern countries. It has been used for centuries as an anthelmintic in man and animals, and it has digestive, astringent, cardiotonic, and emmenagogic properties. The refining processes for the fruit, the chemistry and uses of the husk, and the chemistry of the seed or areca nut proper are described. The pharmaceutical properties have been shown to be due to the predominant alkaloid, arecoline. Synthetic arecoline hydrobromide has been shown to have properties similar to the endogenous material. Chewing areca nuts is very popular in the Eastern countries. Excess chewing is said to give rise to intestinal irritations and diarrhea and may even lead to buccal carcinoma. Combinations with lime, betel leaves, and tobacco (a popular concoction among users) potentiate the carcinogenic effects of the nut. Since oral cancer predominates in India, some type of control of the chewing habit is advocated. (93 references)

3622 ANOTHER FOOD CHEMICAL, AF-2, ALPHA-2-FURYL-5-NITRO-2-FURANACRYLAMIDE. (Eng.) Kennard, C. H. L. (Dept. Chemistry, Univ. Queensland, Australia). *Int Flavours Food Addit* 7(2): 59-60; 1976.

On Sept 1, 1974, the use of the food additive α-2-furyl-5-nitro-2-furanacrylamide (AF-2) was banned in Japan. It had previously been found to be an effective preservative by inhibiting the electron transfer coupled with NADH in the bacterial respiratory chain. However, it was found to be mutagenic to *Escherichia coli*, and evidence indicates it may modify the DNA in mammalian germ cells. It was also found to produce chromosome aberrations in cultured human lymphocytes. The LD_{50} of AF-2 for mice was 0.57 g/kg po. It is not known how AF-2 is metabolized in the body.

3623 SIDE EFFECTS OF ORAL CONTRACEPTIVES: HYPOTHESES AND FACTS. (Fre.) Kuttenn, F. (Clinique Medicale I, Hopital de la Salpetriere, 47, boulevard de l'Hopital, 75634 Paris Cedex 13, France). *Rev Prat* 26(12): 898-900; 1976.

The harmful side effects of oral contraceptives are reviewed. While the original pill had an adequate estrogen-progesterone ratio, most pills today have a marked hyperestrogenic balance (50 µg ethinyl estradiol/l to 5 mg progesterone), dangerous because synthetic estrogens are responsible for the majority of the side effects and also because progesterone deficiency has been recognized as one of the factors in mammary and endometrial cancers. Women on the pill may suffer from any of the following side effects: slowing of the flow of bile, with subsequent hepatic trouble during pregnancy; hyperinsulinism and a curve of diabetic-type hyperglycemia; increase in blood cholesterol; increase in blood pressure; interference with blood coagulation; increased danger of thrombosis; headaches and aggravation of prior epileptic conditions; menstrual troubles, including amenorrhea; and psychological side effects. Clearly such factors as obesity, prior hypertension, and diabetes increase the susceptibility to side effects. A history of mastopathy, which some consider a precancerous state, creates an added danger in some women. Although statistics do not indicate a rise in uterine cancer, the possible carcinogenic potential of synthetic estrogens makes continued vigilance over the side effects of oral contraceptives a necessity. (6 references)

3624 MODIFYING FACTORS IN RAT MAMMARY GLAND CARCINOGENESIS.

(Eng.) Shellabarger, C. J. *In: Biology of Radiation Carcinogenesis*. Edited by J. M. Yuhas, R. W. Tennant, and J. D. Regan. (New York: Raven Press): pp. 31-43; 1976.

The following facts were noted from a review of the literature concerning modifications of rat mammary gland carcinogenesis. The spontaneous incidence of mammary adenocarcinomas and mammary fibroadenomas is related to the strain of rat studied. Rats that are sensitive to chemical carcinogens, with regard to induced mammary neoplasia, tend to be the same strains that are sensitive to radiation. Following methylcholanthrene (MCA) and x-ray or MCA and neutron administration, induction of mammary adenocarcinomas is additive. Lactating and older rats lose responsiveness to chemical carcinogens but not to radiation. When studied over short periods of time: (a) radiation appears to act by a scopal mechanism; that is, most of the mammary neoplasms following exposure are found in the irradiated volume of mammary gland tissue; (b) mammary neoplasia induction was not changed when low linear energy transfer (LET) radiation was split into two equal fractions; (c) high LET radiation is more effective than low in inducing mammary neoplasia. One report suggests that dimethylbenzanthracene can act as an initiator of mammary adenocarcinomas and that phorbol can act as a promotor. Viruses are also reported to participate in mammary neoplasia induction. Radiation and diethylstilbestrol appear to act synergistically in inducing mammary adenocarcinomas in one strain of rat but not in another. (26 references)

3625 BIOLOGIC CHARACTERISTICS OF TUMOR PROGRESSION IN THE MOUSE MAMMARY GLAND.

(Eng.) Medina, D. (Baylor Coll. Medicine, Houston, TX 77025). *Cancer Chemother Rep* [*2*] 5(1): 127-131; 1976.

A new model system for studying the effects of therapeutic drugs and manipulations on the transformation of preneoplastic to neoplastic tumors is proposed. The intermediate stages of various mouse mammary gland tumors were isolated and studied. Hyperplastic alveolar nodules (HANs), the most frequently encountered intermediate preneoplastic nodules, are identified as discrete foci of alveolar hyperplasias in nonpregnant, nonlactating females. HANs can be induced by mammary tumor virus (MTV), by various chemical carcinogens, or by prolonged hormone stimulation. The frequency of tumor formation depends upon the genotype of the nodule transplants, and all HANs are capable of infinite propagation. However, HANs are still controlled by local regulatory factors-tumorigenesis and growth are dependent on anterior pituitary hormones. The characteristics of a resultant tumor are determined by the properties of the cells in the nodule that progress to neoplasia. Noduligenesis and tumorigenesis are inhibited by phenylalanine-deficient diets. It is concluded that the inhibition is due to restricted caloric intake and altered hormonal milieu. This system may offer a model system for study of the intermediate stages of transformation, stages that could be targets for cancer therapy. (21 references)

3626 HORMONE PROFILES IN HORMONE-DEPENDENT CANCERS.

(Eng.) Zumoff, B. (Montefiore Hosp. and Medical Center, Bronx, NY 10467); Fishman, J.; Bradlow, H. L.; Hellman, L. *Cancer Res* 35(11/Part 2): 3365-3373; 1975.

Studies on the relation of urinary excretion of androgen metabolites and estrogens to the natural history of breast cancer are reviewed. The importance of distinguishing between ''within-population'' studies (namely cancer patients versus normal controls) and ''between population'' studies (namely low-risk versus high-risk populations) is emphasized. It is pointed out that ''qualitative'' agreement (the same direction of differences) between the two types of studies must be present in order to implicate a hormonal parameter as a determinant of the natural history of breast cancer. The reported relationship of low urinary androgen metabolite excretion to increased risk of developing breast cancer and poor response to adrenalectomy or hypophysectomy and the validity of the ''estriol hypothesis,'' namely, that a high urinary ratio of estriol to estrone-plus-estradiol in early life is protective against subsequent development of breast cancer, are both dubious. A new hypothesis concerning the relationship of estrogens to breast cancer risk is presented: ''A period of time, prior to age 30, during which the amount or biological availability of active estrogens (namely estrone and estradiol) is diminished, protects against subsequent development of cancer.'' (81 references)

3627 NEONATAL INDUCTION OF HEPATIC AND OTHER TUMOURS. (Eng.) Roe, F. J. C. *In: Mouse Hepatic Neoplasia. Proceedings of a Workshop held at the H.T.S. Management Centre, Lane End, High Wycombe (Great Britain), 12-17 May 1974.* Butler, W. H.; Newberne, P. M., eds. (New York: Elsevier Scientific Publishing Co.): pp. 133-142; 1975.

Neonatal chemical induction of hepatic tumors in mice was reviewed and compared to the spontaneous appearance of these tumors in untreated adult mice. The animals were treated with 7,12-dimethylbenz(a)anthracene (DMBA), 4-dimethylaminoazobenzene or its 2-methyl or 3'-methyl derivatives, and with benz(a)-anthracene, chrysene, 7-methyl(a)anthracene, and their respective K-region epoxides. There was an increase in the multiplicity of liver tumors in the animals that bore them, rather than an increase in the proportion of the mice that developed them, and males were more susceptible than females. The liver tumors seen in young DMBA-treated mice were exactly the same type as those seen in untreated older animals. The histological characteristics of the tumors that arose in response to chemicals given at birth were similar to those of spontaneously occurring tumors. In general, most of the lesions were neoplastic, and a significant proportion, especially in older mice, was malignant. In mouse strains in which there is a 100% incidence of liver tumors in old age, it is difficult to know whether the tumors that arise early are induced by chemical treatment or represent enhancement by another factor. The possibility that other nonspecific mechanisms may be involved should be considered because germ-free status, sex, and dietary intake can affect the age-standardized incidence of liver-cell tumors in otherwise untreated mice. An increased incidence of liver-cell tumors in mice exposed at birth to a chemical compound is therefore not a reliable indication that the compound is carcinogenic. (18 references)

3628 RESPONSE OF HOMOZYGOUS AND HETEROZYGOUS XERODERMA PIGMENTOSUM CELLS TO SEVERAL CHEMICAL AND VIRAL CARCINOGENS. (Eng.) Stich, H. F. *In: Molecular Mechanisms for Repair of DNA: Part B.* Edited by P. C. Hanawalt and R. B. Setlow. (New York: Plenum Press): pp. 773-784; 1975.

The literature concerning various related aspects of cancer and DNA alterations and their repair is reviewed. The aspects are: the relationship between the carcinogenicity of various compounds and their capacity to elicit DNA damage that results in a DNA repair synthesis; the metabolic activation of precarcinogens; the in vivo use of DNA repair synthesis to identify tissues that will give rise to neoplasms; the possible involvement of DNA repair in the sensitivity of human cells to the chromosome-damaging and lethal effect of chemical carcinogens; the different responses of homozygous xeroderma pigmentosum (XP) cells, heterozygous XP cells, and controls to some but not all chemical carcinogens and oncogenic viruses; and the possible link between DNA damage, its repair, and viral-induced neoplastic transformation. The data indicate that the extent of DNA repair plays a crucial role in the early stages of neoplastic transformation, when carcinogens interact with the host-cell DNA, and that DNA repair affects the response of cells not only to UV irradiation but also to challenge by chemical carcinogens and oncogenic viruses. (36 references)

3629 PATHOGENESIS OF MAREK'S DISEASE. (Eng.) Payne, L. N. (Houghton Poultry Res. Station, Houghton, Huntingdon, Cambs., England); Frazier, J. A.; Powell, P. C. *Int Rev Exp Path* 16: 59-154; 1976.

A comprehensive review is presented of the pathogenesis of Marek's disease, a lymphomatous and neuropathic disease of domestic chickens. It is not common to other birds and is apparently without an exact counterpart in man. The disease is caused by a herpesvirus, and it can be controlled by the use of live virus vaccines based on attenuated virus, an apathogenic herpesvirus of turkeys, or apathogenic strains of virus. Many millions of chickens are now vaccinated every year against the disease, which shows that large-scale vaccination against common virus-induced cancer is possible. Discussion is given of historical concepts and nomenclature; virus-cell relationships; the pathology of natural and experimental transmission of the disease; the acquisition of active and passive immunity; the immunosuppressive effects of the disease; and factors affecting pathogenesis and immunity, such as virus strain, the genetic constitution of the host, and the age and sex of the host. (368 references)

3630 THE EPIDEMIOLOGY OF AVIAN LYMPHOID LEUKOSIS. (Eng.) Crittenden, L. B. (United States Dept. Agriculture, ARS Animal Physiology and Genetics Inst., Beltsville, MD 20705). *Cancer Res* 36(2): 570-573; 1976.

The circumstances surrounding avian viral infection were summarized. Lymphoid leukosis viruses of subgroups A, B, C and D are able to induce avian lymphoid leukosis whereas other subgroups differ in pathogenic potential. The endogenous virus of the chicken (Rous-associated virus type 0) belongs to Subgroup E and has little, if any, potential for inducing lymphoid leukosis. Nearly all chicken flocks are infected with Subgroup A lymphoid leukosis virus. Early infection, either by congential means, or soon after hatching, leads to the highest incidence of lymphoid leukosis. Maternal antibody or genetic resistance to infection delays or prevents infection, leading to a lower incidence of disease. In flocks segregating for genetic resistance to infection, continued infection is maintained through dynamic interactions between genetic resistance, acquired or maternal antibody, and virus infection. Expression of endogenous viral information is controlled by dominant genes but spontaneously produced Rous-associated virus type 0 can spread through a susceptible flock and and be transmitted like an exogenous virus. (21 references)

3631 RELEVANT NEW CONCEPTS: ONCORNAVIRUS TRANSFORMATION. (Eng.) Bauer, H. *In: Comparative Leukemia Research.* Edited by J. Clemmesen and S. D. Yohn. (Basel, Karger): 534-538; 1976.

Evidence of how the Rous sarcoma virus genome causes changes in various cell surface properties was discussed. Indirect evidence exists for the theory of one-gene transformation (''onc'' gene), the candidate for the gene product being a glycoprotein called tumor specific cell surface antigen. The majority of temperature-sensitive (ts) mutants isolated have been defective for transformation only and seem to have defects near the 3' end of the genome. This points to either a mutagenic hot spot or an acutely susceptible protein in this region. Partial defective mutants (PD) have also been isolated that are like conventional ts transformation mutants in their ability to induce experimental tumors. A possible explanation for the two categories of ts transformation is that there must be at least two fundamental transformation functions that are virus-coded. Both would be defective with conventional mutants, with just on being defective with a PD mutant. (5 references)

3632 PHYSIOPATHOLOGY OF HUMAN AND VIRUS-INDUCED MURINE LEUKEMIAS. (Eng.) Jasmin, C. (Institut de Cancerologie et d'Immunogenetique (INSERM), Hopital Paul-Brousse, 14-16, Avenue Paul-Vaillant-Couturier, 94800 Villejuif, France); Joffe, F. S.; Klein, B.; Kerdiles-LeBousse, C. *Cancer Res* 36(2): 603-607; 1976.

Friend leukemia was studied to develop a model for differential leukemias. In Friend leukemia, Friend virus induces permanent differentiation of erythropoietin-responsive cells. This erythropoietic proliferation and maturation is accompanied by a marked cell loss and provokes enlargement of the stem cell compartment. The so-called leukemic cells have a limited proliferation capacity and may not be truly malignant as opposed to blastic cells in acute leukemias. It is suggested that these differentiated leukemias initiate from an uncontrolled differentiation of a committed cell compartment, which stimulates proliferation of the stem cell compartment. The disease would be due to a proliferation and accumulation of ''subnormal'' cells characterized by a shorter mean life-span than the normal differentiated cell population. The available data suggest that the physiopathology of acute leukemias is clearly distinguishable from that of differentiated leukemias. (39 references)

3633 MAPPING THE GENES OF SIMIAN VIRUS 40. (Eng.) Lai, C.-J.; Nathans, D. *In: Tumor Viruses. Cold Spring Harbor Symposia on Quantitative Biology.* Cold Spring Harbor Lab. (Cold Spring Harbor, NY): vol. 39, pp. 53-60; 1975.

The following approaches were used to map simian virus 40 (SV40) genes: (a) temperature-sensitive (ts) mutants were mapped by marker rescue with endonuclease restriction fragments of viral DNA and (b) deletion mutants were constructed and mapped. Both procedures used site-specific restriction endonucleases to provide specific fragments of SV40 DNA. In the marker rescue experiments these fragments were used directly to provide wild-type nucleotide sequences at the site of mutation, thereby correcting the ts defect. The region of the genome containing the mutant site could be identified, as the order of fragments in the SV40 genome is known. In the deletion mutant experiments, genomes lacking a DNA segment were constructed by enzymatic excision and subsequent cloning by complementation with a ts mutant. The three ts A mutants tested to date map in a small region comprising only about one-third or less of the early region. This could reflect the gene size or a peculiar property of the gene product, or it might be fortuitous. Also, ts B and ts C mutants, defective in virion protein(s), map in the same segment of the late region. Both D mutants studied map in *Hemophilus influenzae* restriction enzyme fragment E, a late region; therefore, the D gene product may be a virion protein. (31 references)

3634 EARLY EVENTS IN ADENOVIRUS-INFECTED CELLS. (Eng.) Russell, W. C. *In: Tumor Viruses. Cold Spring Harbor Symposia on Quantitative Biology.* Cold Spring Harbor Lab. (Cold Spring Harbor, NY); vol. 39, pp. 551-557; 1975.

Serological, polypeptide synthesis, and temperature-sensitive mutant studies on adenovirus-infected cells are reviewed. Serological studies suggest that adenovirus-derived tumors have components that are related to both nonstructural and structural (possibly fiber) antigens. Recent evidence suggests that core proteins are synthesized early in infection, and since the synthesis of cellular histones is inhibited later in infection, it is suggested that the basic core polypeptides may play a role in the replication of virus DNA somewhat analogous to that of histones. If infected cell-specific polypeptides and the core polypeptides are associated with the replication of viral DNA, they may be DNA-binding proteins. The early findings that broad tumor sera react type-specifically with fiber antigen can be explained by the possibility that some of the reactions against structural components can be related to antibody against the core components (probably made early in infection), or the possibility that the fiber antigen preparations were not rigorously purified. These studies indicate that only a few possible viral gene products can be detected early in a lytic infection or in an abortive infection. Some or all of these products appear to be concerned with initiating and maintaining viral DNA synthesis; failure to do so may lead to integration of the viral genome into the cellular chromosome. The integrated viral genome would probably express a viral gene function that would kill the host; in a few cases, the functions that are integrated or expressed may be compatible with the coexisting cellular functions and thus could lead to transformation and oncogenesis. (46 references)

3635 THE REPLICATION OF PAPOVAVIRUS AND ADENOVIRUS DNA. (Eng.) Levine, A. J. (Dept. Biochemical Sciences, Princeton Univ., Princeton, NJ, 08540); Van der Vliet, P. C.; Sussenbach, J. S. *Curr Top Microbiol Immun* 73: 67-124; 1976.

Recent developments in the study of SV40, polyoma, and adenovirus DNA replication are reviewed; all three viruses can replicate in the nuclei of monkey, mouse, or human cells. Papovaviruses and adenoviruses have been shown to cause tumors in animals and transform cells in culture. The study of DNA replication is useful since it is an integral part of the lytic infection and is relevant to the understanding of viral transformation and the regulation of viral and cellular gene expression. The unique nature of the origin of SV40 DNA replication suggests that the proteins involved must recognize a unique base sequence or structure in the SV40 chromosome. Adenoviruses are much larger and more complex than papovaviruses, and no evidence exists for the presence of cellular histones in the adenovirus particle, unlike papovaviruses. Specific nucleic acid-protein interactions play an integral role in lytic infection, DNA replication, and possibly transformation of papovaviruses and adenoviruses. The isolation and characterization of early viral functions should yield valuable information on the mechanisms of DNA replication and transformation. (298 references)

3636 IN VITRO TRANSFORMATION OF MAMMALIAN CELLS BY HUMAN HERPES VIRUSES. (Eng.) Rapp, F. (Pennsylvania State Univ., Coll. Medicine, Hershey, PA); Hutt, R. *Adv Pathobiol* 5: 112-135; 1976.

The role of herpesviruses in human neoplasia and the conditions necessary for expression of their oncogenicity are reviewed. There are numerous links between herpesviruses and neoplasia in animals, including nonhuman primates. In addition, Epstein-Barr virus (EBV) is regularly associated with certain human neoplasms (Burkitt's lymphoma, Hodgkin's disease, a poorly differentiated lymphocyte-lymphoblast lymphoma, and nasopharyngeal carcinoma), and it has reproducibly transformed both human and simian cells in vitro, with evidence of continued viral presence. Cells transformed in vivo or in vitro demonstrate chromosomal aberrations and membrane changes that may be necessary for the expression of oncogenicity by herpesviruses. Herpes simplex virus (HSV) type 2 has been seroepidemiologically associated with the etiology of squamous cervical carcinoma, herpesviruses have been observed in malignant cells, and HSV can induce neoplastic disease in animal systems. Four strains of HSV-1 and seven of HSV-2 have shown detectable transforming potential in vitro; the data point to strain resistance, to the requirement for far less than the total genome to effect transformation, and to continued conservation of the viral genome in the transformed cells. Cytomegalovirus (CMV) has been associated with Kaposi's sarcoma, atypical lymphocytes, and leukemia, although it is unknown whether it plays a causative role. CMV can stimulate host cell DNA and RNA synthesis in a variety of in vitro systems, and it produced malignant transformation in hamster embryo fibroblasts. Normal human prostate cells carrying CMV underwent transformation in vitro, confirming the transforming potential of CMV in humans. The question is probably not whether EBV, HSV, or CMV cause human cancer but whether other factors and environmental conditions play a role in favoring such development. (141 references)

3637 CARCINOMA OF THE CERVIX--AN EPIDEMIOLOGICAL PERSPECTIVE. (Eng.) Clarke, M. *In: Gynaecological Malignancy: Clinical and Experimental Studies*. Edited by M. G. Brush; R. W. Taylor. (Baltimore: Williams and Wilkins): pp. 3-15; 1975.

Cancer of the cervix is a relatively uncommon cause of death but it does bring a heavy toll in middle-aged women, accounting for 4% of all deaths in the age group 45-64. Women of the lower socioeconomic classes and women who are divorced or widowed experience higher death rates from the disease than women in the upper social classes and women who never married. The standardized mortality ratios for social classes I to V are 34, 64, 100, 116, and 181, respectively. Other factors associated with cervical cancer include age at first coitus and multiplicity of sex partners. There appears to be a close relationship between herpes simplex virus type 2 and cervical cancer. The screening for cervical cancer by cervical smears remains of unproved effectiveness for reducing mortality. Attendance for smear taking is poor among women of lower socioeconomic status, who are at high risk, and there is considerable disagreement among pathologists on the classification of smear specimens, resulting sometimes in inappropriate treatment. (33 references)

3638 ARE THE HERPES SIMPLEX-GENITALIS VIRUSES A CAUSE OF CERTAIN HUMAN CANCERS? (Eng.) Sabin, A. B. (Medical Univ. South Carolina, Charleston, SC). *Adv Pathobiol* 5: 136-150; 1976.

Relevant existing evidence concerning the etiologic role of the herpes simplex (HSV)-genitalis viruses in human cancers is reviewed, and the most valuable direction for future research is proposed. Cancers experimentally produced by SV40, polyoma virus, and certain adenoviruses are discussed in light of their applicability to tests for a possible role of a comparable DNA virus in certain human cancers. Cancers experimentally produced by HSV-genitalis viruses are also discussed. The two striking differences of the latter from the polyoma-SV40-adenovirus group of transformed cells and tumors are: (a) the relatively high frequency of synthesis of some viral structural antigen(s) in HSV-free cells and tumors; and (b) the failure to find nonstructural T antigen in any of the transformed cells or tumors by immunofluorescence with sera from tumor-bearing hamsters. Approaches previously used to implicate HSV in human neoplasia include: (a) demonstration of differences in the prevalence of HSV-2 neutralizing antibodies in cancer patients and ''matched'' controls; (b) tests for HSV-2-specific thymidine kinase or for hypothetical nonvirion antigens in the cancer tissue or for antibodies against them in patient sera; and (c) biochemical tests for HSV-2 genome or its expression in human tumors. None have provided acceptable evidence that HSV are etiologically involved in any human cancer, although these viruses continue to be suspect. A definitive answer is most likely to be provided by epidemiologically controlled biochemical data showing the consistent presence or absence of transforming, unique sequences of HSV DNA in some varieties of cancer and not in other cancers or in properly selected normal tissues. (69 references)

3639 THE PATHOGENESIS OF LYMPHOMA AND PARALYSIS IN WILD MICE.

(Eng.) Gardner, M. B. (Univ. Southern California Sch. Medicine, 1840 North Soto St., Los Angeles, CA 90033); Klement, V.; Rasheed, S.; Rongey, R. W.; Brown, J. C.; Pike, M.; Henderson, B. E.; Huebner, R. J. *Bibl Haematol (Basel)* (43): 204-208; 1976.

Studies of the pathogenesis of naturally occurring lymphoma and paralysis in wild mice are summarized. The incidence and the age of onset are related to the level of indigenous type C virus expression early in life. Type C virus from two different populations of wild mice in Southern California (LP and LC) causes lymphoma and neurogenic lower extremity paralysis both naturally and experimentally. In mice prone to this disease, the indigenous virus is ecotropic, expressed soon after birth, and is present in high titers in both viscera and sera of most young adult mice. Differences in host immune and other defense mechanisms may greatly influence susceptibility or resistance; a few adult wild mice remain free of disease. The LP and LC viruses constitute a third class of murine lymphoma virus designated as amphotropic because of their wide in vitro host range for mouse (N-tropic) and many heterologous mammalian species. They are probably the ancestral viruses from which ecotropic and xenotropic viruses of laboratory mice have been derived. Viremic exposure to the greatest amount of virus in the first days of life probably accounts for greater risk of disease. Leukemogenesis is associated with relatively little increase in virus titer in LC mice. There is little or no evidence of humoral or cellular immune response to the indigenous infection, nor is there any evidence of viral immune complex deposition or related disease. Radioimmune precipitation detects a viral immune response in those few adult mice free of infectious virus. Immunosuppressive treatment of LC and LP mice does not alter the incidence of lymphoma and paralysis. The general immune responsiveness of LC mice to nonviral antigens is probably normal. Cross-breeding of LC wild mice with C57 B10Sn inbred mice leads to a nearly total suppression of LC virus expression early in life. (18 references)

3640 EPSTEIN-BARR VIRUS (EBV)-DETERMINED CHARACTERISTICS IN LYMPHOID CELLS.

(Eng.) Klein, E. (Dept. Tumor Biology, Karolinska Institutet, S-104 01 Stockholm 60, Sweden); Neumann, H.; Steinitz, M.; Yefenof, E. *Bibl Haematol (Basel)* (43): 13-18; 1976.

The characteristics of lymphoid cells infected with Epstein-Barr virus (EBV) that are attributed to the infection are reviewed. A 14-translocation may be responsible for promoting transformation to malignant growth in Burkitt's lymphoma. All EBV genome-carrying cell lines, regardless of viral production, carry a nuclear antigen, EBNA. In virus-producing lines, several other cell-membrane antigens are also present. No antigenic differences have been detected between the two known biological virus variants, P3HR-1 and B95-8, but the DNA of the two appears to be different. Membrane-associated IgM capped more readily in three EBV-negative lines than in EBV-positive lines. Although EBV-positive cell lines of nonmalignant derivation occasionally grow in nude mice, Burkitt lymphoma lines appear to grow more readily. The viral genome also causes differences in temperature sensitivity. Alkaline phosphatase from Burkitt-derived lymphoblastic cell lines appears to be different from normal alkaline phosphatase in terms of substrate specificity and electrophoretic mobility. Patients with infectious mononucleosis also have this altered alkaline phosphatase, called N-apase. (31 references)

3641 EVIDENCE FOR INFECTIOUS COMPONENT OF HODGKIN'S DISEASE AND RELATED CONSIDERATIONS.

(Eng.) Vianna, N. J. (Cancer Control Bureau, New York State Dept. Health, Albany, NY 12237). *Cancer Res* 36(2): 663-666; 1976.

The following four epidemiological approaches used to evaluate the hypothesis that Hodgkin's disease might have an infectious component were summarized: (a) the possibility that prior tonsillectomy might predispose to Hodgkin's disease, (b) studies of case groupings, (c) occupational mortality studies, and (d) family studies of this disorder. Evidence suggests that the cellular surface of thymus-derived lymphocytes undergoes some antigenic alterations during viral infection. Hodgkin's disease might then be viewed as the result of a chronic immune reaction, characterized by an eventual depletion of T-cells, caused by viral infection of these cells and by normal T-cells interacting against antigenically altered ones. The hypothesis gains additional plausibility by the fact that it might explain the heterogeneity of cells (for example, lymphocytes, plasma cells, and eosinophils) found in typical Hodgkin's disease lesions, the axial lymphatic distribution of Hodgkin's disease, and the occasional presence of anergy. It is postulated that a variety of factors, both environmental and genetic, might be important in the etiology of this disorder. (35 references)

3642 DEVELOPMENTS AND TRENDS IN TUMOR VIROLOGY.

(Eng.) zur Hausen, H. (Insitut fuer klinische Virologie, Universitaet Erlangen-Nuernberg, 852 Erlangen, West Germany). *Adv Biosci* 12: 561-566; 1974.

Three problems in tumor virology are considered: (a) the mechanism that underlies the oncogenic action of tumor viruses; (b) the search for human tumor viruses and their relationship to spontaneously occurring human tumors; and (c) the development of a specific chemotherapy and useful vaccines. The DNA of SV40 and polyoma viruses becomes covalently linked in vitro with host cell DNA, and virus production can be induced by fusing the nonpermissive transformed cells with permissive cells. Linear human adenovirus DNA becomes covalently linked to host cell DNA after extensive fragmentation, and it is impossible to induce the synthesis of infectious virus in cells transformed by these agents. Virus-specific DNA has also been discovered in cells transformed by RNA tumor viruses. Viral RNA is transcribed into DNA after infection by the reverse transcriptase present within the virion. Except for RNA-containing tumor viruses, genomes of DNA viruses are only partially transcribed within transformed cells. This transcription leads to the production of T antigens that are virus specific and present in most virally transformed cells. Transplantation specific tumor antigen may also play an important role in the change of growth properties and the mobility of the transformed cells. The mechanism of transformation remains unresolved. Epstein-Barr virus (EBV) is thought to be the causative agent of Burkitt's lymphoma and possibly also of nasopharyngeal carcinoma, and herpes simplex type 2 has been implicated in the induction of human cervical cancer. Another human herpesvirus, the cytomegalovirus, has been considered in relation to Kaposi's sarcoma, but the evidence is still far from convincing. Human wart virus is suspected of producing papillomas of the skin, but recent experiments failed to demonstrate wart viral DNA in condylomata acuminata, papillomas of the larynx, and in some verrucae vulgares. Very little is known of the prevention and chemotherapy of infections with oncogenic viruses. Specific inhibition of reverse transcriptase by rifampicin could indicate the beginning of the development of a chemotherapy aimed specifically at tumor viruses. (No references)

3643 TYPE-C VIRUSES AND LEUKEMIA. (Eng.) Gallo, R. C. (Lab. Tumor Cell Biology, Natl. Cancer Inst., Bethesda, MD 20014). *Adv Pathobiol* 4: 47-66; 1976.

The relationship between type C viruses and leukemia is reviewed. Problems complicating the proof that these viruses cause leukemia derive from the long latency period, congenital and vertical transmission, the variety of properties of these viruses, and the difficulty in fulfilling Koch's postulates, even in animals. A considerable body of information is available regarding the biochemistry and replication of this group of viruses. Most of the endogenous C viruses are not tumorigenic, but most of those that contain an RNA genome with nucleotide sequences that are detectably different from the DNA of the normal host cell are oncogenic. Both types are present in primates. Reverse transcriptase, viral-related nucleic acid, and p30 protein have been identified in the peripheral blood of human myelogenous leukemia patients, and these components were specifically related to components of primate type C RNA tumor viruses. A factor from a whole human embryo cell line has been identified that promotes exponential growth and differentiation of human myelogenous leukemia cells in liquid suspension tissue culture. The relevance of this to the pathogenesis of leukemia is discussed. (62 references)

3644 VIRUSES AND OTHER MICROBES. (Eng.) Heath, C. W., Jr.; Caldwell, G. G.; Feorino, P. C. *In: Persons at High Risk of Cancer. An Approach to Cancer Etiology and Control.* Edited by J. F. Fraumeni, Jr. (New York: Academic Press, Inc.): pp. 241-265; 1975.

The role of viruses, bacteria, and parasites in carcinogenesis is reviewed. Although there is no absolute proof of the oncogenicity of Epstein-Barr virus, considerable circumstantial evidence has been assembled, particularly in relation to Burkitt's lymphoma, nasopharyngeal carcinoma, and, to a lesser degree, Hodgkin's disease and other lymphoproliferative tumors. Evidence also suggests that herpes simplex virus type (HSV-2) predisposes to cancer of the uterine cervix, but conclusive proof is lacking. Retrospective case-control serologic surveys revealed a fairly strong and consistent association between the presence of anti-HSV-2 antibodies and cervical cancer. The association appeared to hold true for earlier stages of carcinoma in situ and dysplasia as well. It has been hypothesized that varicella infection during pregnancy may heighten the risk of cancer in offspring. There is little evidence that infection with human RNA or DNA viruses other than the herpes group increases the risk of cancer. Speculation that simian virus 40 might be related to human tumors first arose when this virus was found in early batches of killed poliomyelitis vaccine. According to retrospective studies, there is an increase in brain tumors among children of women receiving killed polio vaccine during gestation and an increased risk of cancer in children immunized against poliomyelitis. Indirect observations have related intestinal bacterial flora to colon cancer and possibly to breast cancer and urinary tract infection to gastric cancer. Relationships between parasitic infection and cancer risk have been suggested for malaria, schistosomiasis, and infection by the oriental liver fluke, *Clonorchis sinensis*. Little or no evidence exists for epidemicity, that contact with cancer patients increases the risk of cancer, or that viral infection is a cause of familial cancer. (232 references)

3645 PERSPECTIVES IN CANCER RESEARCH. ONCOGENIC VIRUSES: IMPLICATIONS FOR HUMAN DISEASES (PART II). (Eng.) Prevost, J. M. (Service de Medecine et Laboratoire d'Investigation Clinique, Institut Jules Bordet, 1000 Brussels, Belgium). *Eur J Cancer* 12(7): 499-510; 1976.

At present, the relationship between viruses and human cancer is largely circumstantial. Among DNA viruses, the Epstein-Barr virus (EBV) is a leading candidate for a human tumor virus. EBV is recognized as the cause of a nonmalignant, self-limited proliferation of B lymphocytes, infectious mononucleosis with Paul Bunnel heterophile antibodies, and is strongly implicated as a cause of Burkitt's lymphoma and nasopharyngeal carcinoma. RNA tumor viruses are known to cause sarcomas and leukemia-lymphomas in a variety of animals and to cause mammary cancer in mice. Evidence is now accumulating that such viruses are indeed present in man and in some human tumors. These putative human viruses are somewhat related to their animal counterparts. The research strategies of various investigators are discussed at length. It is stressed that the recognition of transformation-related and virogenic functions is acutely needed in order to be able to use tumor-specific virus-directed tests. Also, research directed at the biology of the tumor and virus-associated antigens is needed to resolve arguments on the etiology of the disease. (100 references)

3646 STRUCTURAL PROTEINS OF RNA TUMOR VIRUSES AS PROBES FOR VIRAL GENE EXPRESSION. (Eng.)

Strand, M.; August, J. T. *In: Tumor Viruses. Cold Spring Harbor Symposia on Quantitative Biology.* Cold Spring Harbor Lab. (Cold Spring Harbor, NY): vol. 39, pp. 1109-1116; 1975.

The purification and characterization of proteins of type C viruses and their use in the radioimmunoassay as probes for specific viral gene expression are reviewed. Purified virions of RNA type C viruses contain a large number of polypeptides, the chief component being the major internal protein of approximately 27,000-30,000 molecular wt. Other major proteins are the gp69/71, p15, p12, and p10. Purification of Rauscher murine leukemia virus (MuLV) gp69/71, p30, and p15 has been achieved with approximately 50%, 60%, and 40% overall yield, respectively. The purified gp69/71 fraction contains small amounts of carbohydrate residues; both components stained with the Schiff-periodate reagent as glycopeptides, indicating that these polypeptides comprise glycoprotein components of the virus. Gp69/71, is localized in the viral envelope, and both rabbit and goat monospecific antisera to gp69/71 showed strong virus neutralizing activity. Gp69/71, p30, and p15 contain type, group, and interspecies determinants. The relative immunogenicity of the interspecies determinants of the gp69/71, p30, and p15 proteins common to MuLV and feline leukemia virus (FeLV) was analyzed by radioimmunoassay of the precipitation of each by anti-FeLV serum. The antibody titer of this serum was greatest for p30, followed by gp69/71 and p15. Strong evidence indicates viral coding of all three proteins. The expression of gp69/71 and p30 appears to be noncoordinate, and a close linkage between the Kirsten murine sarcoma virus gene(s) and the gp69/71 gene, but not the p30 gene, is suggested. There appears to be close linkage between p30 and gp69/71 structural proteins genes and the GIX trait. The pathogenesis of immune-complex glomerulonephritis in mice may be in some manner related specifically to the expression of the viral envelope glycoproteins and the host immune response to gp69/71. (38 references)

3647 RNA TUMOR VIRUSES AND HUMAN CANCER. (Eng.)

Hehlmann, R. (Medizinische Universitaets-poliklinik, Pettenkoferstr. 8A, D-8000 Muenchen 2, West Germany). *Curr Top Microbiol Immunol* 73: 141-215; 1976.

Present knowledge of RNA tumor viruses as it relates to their detection in human tumors is summarized, and the associations of these viruses with human cancer and human carcinogenesis are discussed. RNA tumor viruses have long been known to cause a variety of animal malignancies, and several examples are cited. Electron microscopy studies are briefly described, and the physical and chemical properties of the viruses are discussed in terms of RNA, protein, enzyme activity, and nucleic acid relatedness. Studies of antigenicity and host range deal with type-, group-, and species-specificity as well as specificity of reverse transcriptase. Replication of RNA tumor viruses is discussed in terms of RNA-directed DNA synthesis and the reaction product. RNA-directed DNA polymerase is present not only in viruses but in apparently noninfected cells. There is electron microscopic evidence for the association of RNA tumor viruses with human leukemias and lymphomas, and suggestive evidence has been obtained from studies of the milk of breast cancer patients. There is also supporting serological evidence for this association, and reverse transcriptase has been detected in human leukemic cells and in human milk. The presence of information specifically homologous to some viral RNA sequences causing analogous diseases in mice and primates has been implied in 48%-91% of human leukemias, 69%-75% of human lymphomas and sarcomas, and 30%-67% of human breast cancers. Simultaneous detection of reverse transcriptase and a high molecular wt RNA in human malignancies is further evidence for the presence of RNA viruses in human tumors. Viral oncogenesis and the origin of RNA tumor viruses are discussed in terms of the provirus theory, the oncogene-virogene theory, the protovirus theory, RNA paraprocessing, endogenous virus, and evolution of the RNA tumor viruses. (650 references)

3648 PERSPECTIVES IN CANCER RESEARCH. ONCOGENIC VIRUSES: IMPLICATIONS FOR HUMAN DISEASES (PART I). (Eng.)

Prevost, J. M. (Institut Jules Bordet, 1000 Brussels, Belgium). *Eur J Cancer* 12(5): 327-340; 1976.

A review of experimental approaches in searching for potential animal and human tumor viruses and current evidence for the role of viruses in human cancer are presented. The general morphologic, biophysical, and biochemical properties of RNA tumor viruses are known and are useful in studying newly isolated viruses. Studies with RNA oncornaviruses indicate that viral genetic information is constantly present in the host cell, probably in the chromosomes. The absolute dependence of viral functions on DNA polymerase for replication and transformation has been shown. Two theories that account for the transforming or tumorigenic properties of oncornaviruses are: (a) viral oncogenesis is accomplished by the introduction of new sets of genetic information in the cell, and (b) viral genes reside in the chromosome of every normal cell. DNA containing oncogenic viruses may result in productive infections in permissive cells or a replicative cycle of the virus, or in cell transformation, in which the virus does not multiply and only some functions are expressed. In animals, papovaviruses are associated with sarcomas and solid tumors in nonnatural hosts and with papillomas; polyoma is not implicated in human cancers, but SV40 is associated with warts and possibly other human conditions. Adenoviruses are associated with sarcomas in animals, but are not implicated in human cancers. Herpesviruses are associated in humans with Burkitt's lymphoma, nasopharyngeal carcinomas, *H. simplex*, and cervical cancer. Even if viruses are a main etiological factor in cancer, tumors arise through complex interactions of these agents at the level of the cell, as determined by its genetic makeup. (119 references)

3649 VIRUSES, GENES, AND CANCER. (Eng.) Dosik, H.; Todaro, G. J. *In: Cancer Cenetics.* Edited by H. T. Lynch. (Springfield, IL: Charles C. Thomas): pp. 101-110; 1976.

The relationship of chromosomal abnormalities to increased risk of malignancy and attempts to identify human populations whose fibroblasts demonstrate an increased susceptibility to T antigen production and transformation by simian virus 40 (SV40) in vitro are discussed. Patients with diseases such as Down's syndrome, Fanconi's anemia, and Bloom's syndrome, which involve chromosomal abnormalities, have an increased risk of leukemia. The involvement of genetic factors in the etiology of cancer is also demonstrated by the association of high malignancy with other kinds of genetic disorders, such as primary immunodeficiency diseases, with exposure to ionizing radiation, and with close genetic matching to affected individuals, as with monozygous twins. The susceptibility of fibroblasts to in vitro transformation by SV40 was high when they were taken from individuals with increased risks of malignancy, such as patients with Fanconi's anemia, Down's syndrome, and other trisomies, radiation-exposed individuals, and siblings of leukemia victims. (50 references)

3650 THE ENDOGENOUS ORIGIN AND TRANSMISSION OF RNA VIRAL GENOMES THAT CODE FOR CANCER. (Eng.) Huebner, R. J. *In: VIth International Symposium of the Association for the Control of Cancer Nordrhein-Westfalen e.V. Dusseldorf October 17-18, 1974.* Swiss League against Cancer. (Dusseldorf, W. Germany): pp. 63-91; 1976.

The presence of endogenous RNA viral genomes that code for cancer, and their role in the natural and experimental induction of cancer in animals, is reviewed. Type C virus genomes are universally present in several mammalian and avian species. Transmission is largely genetic, although horizontal transmission can also take place. Tumors are produced both naturally and experimentally, when the genetic factors controlling their suppression are altered and viral expression results. Virus-specific virogenes used as vaccines have suppressed natural expressions of the virogenes when given prior to high-level natural expressions. Prevention of certain mouse cancers appears to be possible through application of this information. Should type C viruses be isolated as causative agents in human cancer, vaccination against such tumors might be possible. (132 references)

3651 INTERACTIONS BETWEEN LYMPHOCYTES AND C-TYPE ONCORNAVIRUSES IN MICE. (Eng.) Hirsch, M. S. (Massachusetts General Hosp., Harvard Medical Sch., Boston, MA). *Isr J Med Sci* 12(4-5): 407-413; 1976.

Several aspects of type C oncornavirus-lymphocyte interactions in the mouse are reviewed. Various C viruses can induce marked and prolonged depression in the immune responsiveness to diverse antigens. These depressions may involve antibody formation or cell-mediated immunity. The Friend leukemia virus complex has been shown to alter both the organ distribution and cell-surface receptors on B lymphocytes. The degree of immunosuppression is related to the susceptibility of various strains of mice to the leukemia. Virus-induced autoimmunity can also result from infection of mice with exogenous C oncornaviruses. These mice develop both humoral and cell-mediated responses to viral antigens; however, they also develop disseminated lymphomas of thymic origin and usually die at 4-6 mo of age. Considerable evidence suggests that most murine cells contain the necessary genetic information to code for the production of C oncornaviruses. Viral activation can be stimulated by chemicals, radiation, and certain immunological reactions. The host cells have the capacity to respond immunologically to antigens on C viruses. If these responses are ineffective, fully infectious C viruses can be released from the cells. These viruses can infect a variety of lymphoid cells, interfere with their functions, and eventually lead to lymphoid cell tumors. Ideas are discussed for further investigations, such as the study of human immunological responses. (45 references)

3652 EXPERIMENTAL MODELS FOR DNA-RNA VIRAL INTERACTIONS: A BRIEF REVIEW. (Eng.) Frankel, J. W. (Life Sciences Res. Labs., Life Sciences, Inc., St. Petersburg, FL 33710). *Cancer Res* 36(2): 670-673; 1976.

Experiments that demonstrated or implied the occurrence of DNA-RNA viral interactions in man and animals were summarized. The concurrent presence of two dissimilar genetic viruses influenced responses both in vivo and in vitro. Cell-mediated immune responses to Marek's disease herpesvirus were altered in specific-pathogen free chickens inoculated as embryos with an avian leukosis virus. More research is needed to clarify interactions between viral and immunological factors in tumor development. (44 references)

3653 IMMUNE RESPONSE TO LEUKEMIA VIRUS AND TUMOR-ASSOCIATED ANTIGENS IN CATS. (Eng.) Essex, M. (Dept. Microbiology, Harvard Univ. Sch. Public Health, Boston, MA 02115); Sliski, A.; Hardy, W. D., Jr.; Cotter, S. M. *Cancer Res* 36(2): 640-645; 1976.

Developments in the study of various feline carcinomas (spontaneous leukemia, lymphoma, and fibrosarcoma) were summarized. The caustive agents, the feline leukemia and sarcoma viruses, have been well characterized. A specific tumor cell membrane antigen, designated the feline oncornavirus-associated cell membrane antigen (FOCMA) has also been described. Feline leukemia and feline sarcoma viruses are antigenically indistinguishable, and FOCMA is common for both. A clear correlation has been shown between the resistance of cats to development of lethal tumors following inoculation of feline sarcoma virus and the presence of high humoral antibody titers to FOCMA. The geometric mean antibody titer to FOCMA for cats that resisted growth of fibrosarcomas was more than 20-fold higher than the mean for cats that succumbed to lethally progressing tumors. Cats with induced or spontaneous leukemia or lymphoma also have either no detectable FOCMA antibody or very low levels. Conversely, some cats resist development of leukemia or lymphoma following natural exposure to feline leukemia virus in leukemia cluster households, and these cats have high FOCMA antibody titers. These results support the concept of a natural immunosurveillance mechanism against leukemia or lymphoma development in an outbred mammalian species. (65 references)

3654 IMMUNOLOGY--VIROLOGY: GENERAL OVERVIEW. (Eng.) Trentin, J. J. *In: Host Environment Interactions in the Etiology of Cancer in Man. Proceedings of a Meeting Held at Primosten, Yugoslavia, 27 August-2 September 1972.* International Agency for Res. on Cancer and John E. Fogarty International Center of the Natl. Insts. of Health USA. (Lyon, France): pp. 257-263; 1973.

The role of cancer-specific antigens demonstrated in many spontaneous and induced cancers of both man and animals is reviewed. Common tumor antigens in virus-induced tumors constitute an important indicator of the etiology of the tumor. Although these antigens are usually not involved in cancer immunity, there are certain cancer-specific membrane antigens that elicit transplantation immunity; these occur in most spontaneous tumors and in those induced by viruses, chemicals, or physical agents. This group of antigens constitutes the basis for the immunological surveillance defense against cancer. In vivo immunological ''tolerance'' to tumors appears not to represent true immunological unresponsiveness, but rather a blockage, by serum antibody, of cell-mediated immunity demonstrable in vitro. At least some cancer-specific antigens appear to be early fetal antigens that reappear in the tumor, perhaps through a process of genetic derepression. Although viruses are known to cause many animal tumors, a viral etiology has not been proven for any human tumors. However, some serious candidates for human tumor viruses include Epstein-Barr, herpesviruses, herpes simplex type 2, and types B and C RNA viruses. (64 references)

3655 OCCURRENCE OF MALIGNANCY IN IMMUNODEFICIENCY DISEASE. (Eng.) Gatti, R. A.; Good, R. A. *In: Cancer Genetics.* Edited by H. T. Lynch. (Springfield, Ill: Charles C. Thomas): 63-75; 1976.

A review of the literature concerning the incidence of malignancy in some patients with primary immunodeficiencies reveals that the incidence is about 10,000 times that of the general age-matched population. Each type of immunodeficiency has a distinctive constellation of malignancies associated with it. Studies have demonstrated both immunologically aggressive lymphocytes and the presence of blocking antibodies or antigen-antibody complexes in the blood of neuroblastoma patients; therefore, a major role for immunity in oncogenesis seems almost certain. However, the unusual preponderance of lymphoid malignancies among immunodeficient patients suggests that factors other than a faulty immunosurveillance mechanism are involved. (129 references)

3656 IMMUNE DEFICIENCY DISEASES. (Eng.) Kersey, J. H.; Spector, B. D. *In: Persons at High Risk of Cancer. An Approach to Cancer Etiology and Control.* Edited by J. F. Fraumeni, Jr. (New York: Academic Press, Inc.): pp. 55-67; 1975.

The relationship between immune deficiency diseases and malignancy is reviewed. Most of the primary immunodeficiency syndromes associated with increased cancer risk are combined immunodeficiency diseases involving components of both T and B cell systems. The age distribution of cancer in patients with these disorders largely reflects the age at onset of the primary immunodeficiency disorders, so that malignancies are seen mainly in children. The malignancies arise in the following proportions: epithelial, 20%; lymphoreticular, 55%; leukemias, 17%; mesenchymal tumors, 3%; and CNS tumors, 5%. This distribution differs significantly from the pattern of age-matched cases with malignancy in the general population. Data from experimental animals and humans indicate that immunodeficiency predisposes to cancer. In humans, lymphoreticular neoplasms predominate in both primary and drug induced immunodeficiency; the possible causes include: (a) increased malignant transformation of lymphoid cells as a result of intrinsic defects (e.g., chromosomal breaks) or increased activity of endogenous and exogenous oncogenic viruses; and (b) decreased activity of the lymphoid system to recognize and destroy malignant lymphoid cells, as a result of defective recognition or defective response following recognition. A major function of the immune system may be to provide surveillance against malignant cells; according to this hypothesis, in immunodeficiency diseases, the risk of malignancies will increase but the distribution by histologic type will remain unchanged. In patients with both primary and drug-induced immunodeficiency associated with renal transplantation, however, the distribution of malignancies is different from that of the general population. Special attention in future research should be given to the influence of immune responses linked to histocompatibility antigens, since these responses are important in determining cancer susceptibility in mice. (47 references)

3657 MORPHOLOGIC STUDIES OF CELL RECEPTORS OF LYMPHOCYTES AND LYMPHOMAS. (Eng.) Ioachim, H. L. (Lenox Hill Hosp., New York, NY 10021); Schmidt, E. C.; Keller, S. E. *Bibl Haematol (Basel)* (43): 29-33; 1976.

Some of the functional and morphologic characteristics used to differentiate B and T lymphocytes are reviewed, and attempts to identify the cellular origin of various leukemias and lymphomas are discussed. Direct visualization of antibodies on the surface of both benign and malignant B lymphocytes has been possible by immunoelectron microscopy using horseradish peroxidase-labeled anti-IgG and anti-IgM antisera. Lymphoblastic and lymphocytic lymphomas, both nodular and diffuse, are in most cases composed of neoplastic B cells, as are the vast majority of chronic lymphocytic leukemias, Burkitt's lymphoma, and Waldenstrom's macroglobulinemia. Both T and B types have been identified in Hodgkin's disease and, in addition, IgM-labeled Reed-Sternberg cells have been found. Surface marker techniques have allowed recognition of a new clinical-pathologic entity, the T cell lymphoma, a rare neoplasm in children and young adults characterized by localization in the mediastinum, short onset, and rapid and relentless course. (24 references)

3658 CELL-SURFACE TUMOR-ASSOCIATED ANTIGENS OF CHEMICALLY INDUCED TUMORS. (Eng.) Leonard, E. J. *In: Cellular Membranes and Tumor Cell Behavior. Annual Symposium on Fundamental Cancer Research, 1975.* (Baltimore: Williams and Wilkins Co.): pp. 385-397; 1975.

The purification and characterization of a tumor-specific transplantation antigen (TSTA) on line 10 guinea pig hepatoma cells are reviewed. TSTA from line 10 guinea pig hepatoma cells was assayed by several tests, including delayed cutaneous hypersensitivity and antibody absorption tests. The antigen was extracted from line 10 cells by KCl and chromatographed on Sephadex gel and DEAE. The results indicate that the antigen is soluble and heterogeneous, but the yield is low. Antigenicity, measured by the capacity to elicit a delayed cutaneous hypersensitivity reaction in an immune guinea pig, was compared to immunogenicity as measured by tumor rejection. The results show that 700 times more antigen in the form of KCl extract is required to equal the effect of viable cells. A similar or greater discrepancy existed for the line 10 ghosts, despite the fact that the line 10 antigen still resided in undiminished amount in the ghost membrane structure. The great difference between antigenicity and capacity to protect against tumor challenge may indicate that the two tests detect different antigens, or the difference may be a result of an alteration in the antigen quality, continuing antigen production by viable cells, or differences in mode of presentation to antigen-reactive cells. There is still hope that purified tumor antigens may be used to detect, quantify, and enhance specific tumor immunity in patients. (17 references)

3659 SUNLIGHT. (Eng.) Daniels, F. *In: Cancer Epidemiology and Prevention. Current Concepts*. Edited by D. Schottenfeld. (Springfield: Charles C. Thomas): pp. 126-152; 1975.

The small fraction of the sun's radiation between 290 and 320 nm is responsible for the production of skin cancer. White-skinned people are those most prone, especially on exposed areas, and particularly if they work outdoors. The more common types of cancer are basal cell or adnexal cell carcinoma, squamous cell carcinoma, malignant melanoma, and keratoacanthoma. In various parts of the world, the incidence has been increasing. In the US, the incidence is higher in the South than in the North. Queensland, Australia, located near the equator, has the highest incidence in the world. The importance of UV radiation in the etiology of squamous cell cancers is straightforward, but it is more complicated in malignant melanoma. In a Queensland study, no relationship was found with skin color, tanning, or ease of sunburning; thus there seems to be some indirect effect of sunlight on melanoma. There does seem to be a relation with latitude. Scandinavian countries report an increase in mortality as a result of malignant melanoma. In Japan, there is more skin cancer and lupus erythematosus in the south, the area with greater UV radiation. Studies of populations in Hawaii, Singapore, and New Guinea support the idea that skin melanin is a factor in the susceptibility to skin cancers. Both rats and mice are susceptible to UV carcinogenesis; in the latter, one exposure may initiate the tumor, and the growth rate is increased by each exposure. Animal studies relating the type and extent of skin injury with UV wavelength demonstrated damage to both dermis and epidermis; both sites may play a role in human carcinogenesis. Maximum UV radiation is concentrated around solar noon, which should be considered in the prevention and prophylaxis of the disease. To understand cancer risk to individuals, information on clothing, daily activities, and surrounding reflectors and absorbers in the environment should be known. The carcinogenic or cocarcinogenic potential of a reduction in atmospheric ozone with a corresponding increase in UV radiation and of the many newly introduced photosensitizing chemicals is unknown, but may be serious enough to require great care and study. (128 references)

3660 RADIATION. (Eng.) Jablon, S. *In: Persons at High Risk of Cancer. An Approach to Cancer Etiology and Control.* Edited by J. F. Fraumeni, Jr. (New York: Academic Press, Inc.): pp. 151-165; 1975.

The role of ionizing and nonionizing radiation in carcinogenesis is reviewed. Whole body ionizing radiation is responsible for the increased risks of leukemia experienced by physicians, especially radiologists, and the Japanese atomic-bomb survivors. Among the survivors, two-thirds of the leukemias were acute and the remainder were chronic myeloid. In spondylitis patients who received radiation therapy (14,000 cases), mortality from leukemia was nearly 10 times the expected rate. Among these cases, over 80% were acute leukemias and most of the remainder, chronic myeloid. Smaller therapy series showing an increased leukemia risk include women treated for menorrhagia, children irradiated for enlarged thymus glands, and children treated for tinea capitis. An increased leukemia risk has also been seen in thyroid carcinoma patients treated with ^{131}I, in polycythemia vera patients treated with ^{32}P, and in patients injected with thorium dioxide. Risk estimates for leukemia indicate that for children there are about 3 cases of leukemia induced per million person-year-rads over a 20- to 25-yr followup period; for adults the corresponding figure is 1-1.5. Among the Japanese atomic-bomb survivors, excessive mortality from cancer (excluding leukemia) was observed beginning about 15 yr after exposure among persons who received more than 100 rads. The cancers that occurred among exposed children included widely metastasizing thyroid tumors, brain and salivary gland tumors, and cancers of the gastrointestinal system. Among adults, the excess cancer mortality was caused by respiratory cancers, breast cancer, cancer of the gastrointestinal system (excluding the stomach), and lymphosarcoma. Among the spondylitis patients, excesses other than leukemia were seen for cancers of the pharynx, pancrease, bronchi, and lymphatic and hemopoietic systems. Children irradiated as fetuses have a nearly twofold risk of dying from leukemia or any other cancer before age 10. UV radiation is associated with skin cancer; microwave radiation has not been shown to induce cancer in mammals. Research should exploit the differences in energy distribution among the various kinds of radiation to learn about the probable sizes of targets in the intracellular carcinogenic event induced by radiation. (52 references)

3661 MICRODOSIMETRY AND ITS IMPLICATION FOR THE PRIMARY PROCESSES IN RADIATION CARCINOGENESIS. (Eng.) Kellerer, A. M. *In: Biology of Radiation Carcinogenesis*. Edited by J. M. Yuhas; R. W. Tennant; J. D. Regan. (New York: Raven Press): pp. 1-12; 1976.

The concepts of absorbed dose, relative biological effectiveness (RBE), and the time factor are discussed. These concepts are used to quantitate the biological effect of doses of ionizing radiation and are relevant to the mechanism of radiation carcinogenesis. The term ''absorbed dose'' has little meaning when of small numerical value and when small cellular regions are being considered because of statistical fluctuations in energy deposition on a microscopic scale. Criteria have been given to indicate whether these fluctuations are important for a given site diameter and absorbed dose value. At very small doses of ionizing radiation all cellular dose-effect relations must be linear, but at higher doses a quadratic term in absorbed dose becomes significant and results in nonlinearity. When a nonlinear effect is associated with small dose values, as in the induction of mammary tumors in the Sprague-Dawley rat by neutrons, the effect must not reflect damage to independent cells, but an interdependence of some kind between damaged cells. The statistical nature of energy distribution is responsible for the different RBE of different radiations. It is furthermore responsible for the increase in RBE with decreas- RBE on absorbed dose applies both to cellular effects and to the process of radiation carcinogenesis. The time factor, which is the ratio of absorbed doses that produce the same effect at different irradiation times or dose rates, also depends on absorbed dose, and it is meaningless to quote its value without first specifying the absorbed dose. (17 references)

3662 RADIATION-INDUCED STRAND BREAKS IN THE DNA OF MAMMALIAN CELLS. (Eng.) Ormerod, M. G. *In: Biology of Radiation Carcinogenesis*. Edited by J. M. Yuhas; R. W. Tennant; J. D. Regan. (New York: Raven Press): pp. 67-92; 1976.

The occurrence of strand breaks in the DNA of mammalian cells after exposure to ionizing radiation is reviewed. Discussion is made of the experimental methods employed and the analysis of data: the efficiency of production of single-stranded breaks and alternative methods for studying the repair of such breaks; and the anomalous sedimentation of DNA of high molecular wt. The widespread existence of the necessary enzymes and the efficiency of the repair reaction in a variety of different cells indicate that the majority of single-stranded breaks induced by radiation are not in themselves lethal. The same may be true for double-stranded breaks, but further evidence is needed. Recent work has demonstrated that some mammalian cells have the ability to excise certain radiation products of thymine from their DNA. (94 references)

3663 IN VITRO CELL TRANSFORMATION BY LOW DOSES OF X-IRRADIATION AND NEUTRONS. (Eng.) Borek, C. *In: Biology of Radiation Carcinogenesis*. Edited by J. M. Yuhas; R. W. Tennant; J. D. Regan. (New York: Raven Press): pp. 309-326; 1976.

Low doses of x-rays and neutrons, 1-600 rads for x-rays and 0.1-150 rads for 430-keV neutrons, induced neoplastic transformation (as judged by colony morphology) in short term cultures of diploid hamster embryo cells that did not spontaneously undergo transformation. The frequency of transformation was found to be independent of whether the cells were plated singly or at high density in mass culture, whether feeder cells were present or not, or whether the medium was replaced before or after irradiation. However, if the cells were incubated as stationary cultures at 37 C for more than 2 days after irradiation, the transformed cells reverted to normal. This reversion did not occur when the same cultures were incubated at 24 C for 5 days, suggesting that the error-correcting mechanism, which may be active before the cells are able to replicate, is inhibited at 24 C. The ability of in vitro transformed cells to produce tumors in vivo varied among the cell lines studied. In all cases, tumors were fibrosarcomas. The transformed cells were aneuploid with no apparent consistency from one cell to another, but no gross chromosomal aberrations were observed after band staining with quinacrine. The data suggest that those cells within the heterogeneous embryonic cultures most susceptible to transformation by x-irradiation were also most susceptible to the lethal effects of x-rays. Above 300 rads, at which cell death becomes significant, less transformation was observed; two smaller split doses of radiation resulted in increased transformation compared to that obtained after one larger equivalent dose. (26 references)

3664 A MECHANISM FOR THE ENDOCELLULAR CONTROL OF CELL DIFFERENTIATION AND CELL PROLIFERATION. (Eng.) v. Wangenheim, K.-H. (Inst. Botany and Microbiology, Nuclear Res. Centre Juelich, 517 Juelich, West Germany). *J Theor Biol* 59(1): 205-222; 1976.

Evidence in the normal cells of higher multicellular animals of a mechanism that controls the number of mitotic cycles that cell progenies pass through until the various succeeding steps of differentiation are reached and mitotic activity ceases is reviewed, and a control mechanism is outlined. The evidence includes the semiautonomy of cytoplasmic growth, the induction of differentiation by an increase in the cytoplasmic amount:nuclear ploidy ratio, and the suppression of mitotic activity by differentiation. Plant endosperm and radiobiological investigations have also indicated that a quantitative ratio between the number of cytoplasmic factors and the number of chromosome sets per nucleus determines whether a cell remains mitotically active or reaches final differentiation (cell maturation). Cytoplasmic growth during mitotic delay is a prerequisite for radiation-induced early differentiation. A model is proposed to explain the coordination of nuclear and mitochondrial division cycles. It is based on the fact that more than 90% of the mitochondrial proteins are coded for by nuclear genes and synthesized on cytoplasmic ribosomes. It is assumed that mitochondrial repressors control the biosynthesis not only of mitochondrial enzymes, but also enzymes essential for the host cell; for example, those involved in nuclear DNA synthesis and differentiation. It is conceivable that mitochondria are the autoreduplicating cytoplasmic constituent that is essential for the control of mitotic activity and differentiation. Plant plastids may act similarly. (77 references)

3665 EFFECT OF TRAUMA AND METALS ON THE DEVELOPMENT OF TUMORS IN SOFT TISSUES AND BONES.

(Pol.) Witwicki, T. (Oddzial Chirurgii Orazowo-Ortopedyczne, Szpital Bielanski, u.l. Ceglowska 80, 01-809 Warsaw, Poland); Dulowski, W.; Gorecki, A. *Pol Tyg Lek* 31(8): 333-335; 1976.

Among the extraorganic and intraorganic factors in the neoplastic process, trauma and metals occupy a significant position. Trauma in the broad sense is one of the conjectural causes of neoplasms in tissue, but it is still unclear whether the connection between trauma and neoplastic development is causal or coincidental. Studies have confirmed the development of fibrosarcomas and juxtacortical sarcomas secondary to bone fractures, trauma to the chest, or blows from a steel club. Thermic trauma can also cause cancer development. Investigations of the influence of metal prostheses and materials utilized to set broken bones have shown that after a certain period of time, metal introduced into the human system will cause reactions in surrounding tissue. Although the development of a neoplasm around a metal body is rare, untypical reactions promoting the formation of pathological neoplastic tissue are possible, especially in individuals prone to neoplastic growth. This danger has been proved with experimental animals, but there is a lack of convincing evidence for humans.

3666 PRECANCEROUS LESIONS.

(Eng.) Koss, L. G. *In: Persons at High Risk of Cancer. An Approach to Cancer Etiology and Control.* Edited by J. F. Fraumeni, Jr. (New York: Academic Press, Inc): pp. 85-102; 1975.

The precancerous epithelial lesions of various organs were characterized. Those of the uterine cervix form a spectrum of morphologic abnormalities of capricious behavior and unpredictable prognosis. Well-differentiated lesions of squamous epithelium of the type usually observed on the portio of the uterine cervix prevail in the vagina. Teenage girls with adenosis of the vagina are prone to develop vaginal adenocarcinoma. Postradiation in situ carcinoma of the vagina and cervix may occur 1 yr or more after apparently successful treatment of cervical cancer. One form of endometrial hyperplasia, endometrial carcinoma in situ or adenomatous hyperplasia, progresses to endometrial carcinoma in a substantial percentage of cases. Lobular carcinoma in situ develops into homolateral breast cancer after 2-20 yr in 70% of patients and into contralateral breast cancer in 40%. Cigarette smoking is the key epidemiologic factor in bronchogenic epidermoid carcinoma. Epidermoid carcinoma of the bronchus in situ has a very unpredictable prognosis, complicated by the fact that it can produce distant metastases with only minimal infiltration at the primary tumor site. Epidermoid carcinoma in situ forms red velvety patches in the epithelium of the oral cavity that are often mistaken for inflammation; the heavily keratinizing carcinoma of the oral cavity forms white patches that are usually considered benign leukoplakia. Nonpapillary carcinoma of the urinary bladder in situ progresses to invasive carcinoma in 80% of cases over a 5-yr period. Gastric carcinoma follows certain abnormalities of the gastric epithelium such as atrophic gastritis and intestinal metaplasia. Carcinoma of the colon in situ exists either as a flat sessile lesion or as a focal abnormality in adenomatous polyps. Whether all polyps should be considered precancerous is debatable, but in polyposis of the colon, the danger of cancer is very high. Only a small percentage of precancerous skin lesions progress to invasive cancer. Bowen's disease, however, may be associated with systemic cancer in a significant percent and Paget's disease has been associated with an underlying carcinoma of sweat glands. (89 references)

3667 BASIC ASPECTS ON MYCOSIS FUNGOIDES.

(Eng.) Brehmer-Andersson, E. (No affiliation given). *Acta Derm Venereol [Suppl] (Stockh)* 56(75): 22-33; 1976.

The clinical and pathologic features of the three variants of mycosis fungoides, the Alibert-Bazin, the erythroderma, and the d'emblee types, are presented in detail. Mycosis fungoides occurs most frequently in persons from 40-70 yr, with a slightly higher incidence in men. In a study of 144 patients, 7% began with tumors and another 7% with generalized exfoliative erythroderma. The median survival after onset of lesions was 10 yr. In a study of 165 patients, the duration of disease was 8.1 yr for the 106 who died and 12.5 yr for the 59 still alive. Premycotic lesions may appear as erythematous patches or as eczema; bullous lesions are rare. They may be preceded by severe itching, which can continue during the disease, or by prickings or burning pain in the lesions. Tumors may arise in infiltrated plaques, in premycotic lesions, or in apparently normal skin; they have a predilection for the face and body folds, including the axillae, groins, and cubital and breast folds. Itching may diminish or disappear in the tumor stage. Death may result from advanced skin lesions with debility and cachexia, with or without spread to the lymph nodes or internal organs. A leukemoid reaction or lymphocytic or myelogenous leukemias have been reported in a number of terminal cases. No blood changes or cellular changes in the bone marrow characteristic of mycosis fungoides have been regularly reported. (103 references)

3668 MALIGNANT POTENTIAL OF BREAST LESIONS. (Eng.) Dossett, J. A. *In: Risk Factors in Breast Cancer.* Edited by B. A. Stoll. (Chicago: William Heinemann Medical Books Publication): vol. 2, New Aspects of Breast Cancer, pp. 54-66; 1976.

Breast lesions likely to lead to infiltrating cancers are reviewed extensively. The lesion found most closely connected with infiltration is a proliferation of epithelial cells within preexisting boundaries. Ninety percent of infiltrating cancers studied showed evidence of neoplastic proliferative lesions still within their natural boundaries, either within or in direct continuity with the infiltrating cancer. The conclusion is that the majority of breast cancers arise from preexisting, noninfiltrating, neoplastic proliferating cells. Various studies on breast lesions and their malignant potential are reviewed. The results indicate that proliferative lesions, with or without cystic disease, have high malignant potential. The types of proliferative lesions and their grading and anatomical sites are reviewed. The Grade III lesions have the highest infiltration frequency. Less radical approaches to the treatment of breast cancers are advocated, such as aspiration of cysts, biopsy of certain follow-up studies. It is concluded that large-scale studies of breast lesions must be undertaken to develop a more rational approach to breast cancers. (17 references)

3669 THE CEREBELLAR MEDULLOBLASTOMA: ITS ORIGIN, DIFFERENTIATION, MORPHOLOGICAL VARIANTS, AND BIOLOGICAL BEHAVIOR. (Eng.) Rubinstein, L. J. *In: Handbook of Clinical Neurology. Tumours of the Brain and Skull: Part III.* Edited by P. J. Vinken; G. W. Bruyn. (Amsterdam: North-Holland Publishing Co.): vol.18: pp.167-193; 1976.

A review is presented of the occurrence, metastatic spread, and pathological, clinical, cytological, and ultrastructural features of cerebellar medulloblastoma. Because of its histology and clinical incidence it, of all the common gliomas of the CNS, is the one most widely regarded as being truly embryonal. Medulloblastomas account for approximately 6.5%-10% of intracranial gliomas. In children they form a much higher proportion, comprising approximately 25% of all intracranial tumors. The origin of cerebellar medulloblastoma has been attributed to two main sources. The first is either the fetal external layer (the layer of Obersteiner, which gradually disappears during the first 12 mo of life) or its cellular remnants, which have presumably retained their proliferative capacity in later life. The second postulated site of origin, especially of the midline examples, is among those small foci of embryonal cell nests in the germinative zone of the posterior medullary velum. Pigmented papillary medulloblastoma and medullomyoblastoma, which are rare variants of medulloblastomas, are also discussed. (101 references)

3670 THE BIOLOGY OF CHOROID PLEXUS PAPILLOMA AND CARCINOMA OF THE LATERAL VENTRICLE. (Eng.) Laurence, K. M. *In: Tumours of the Brain and Skull, Part II; Handbook of Clinical Neurology.* Vol. 17 Edited by P. J. Vinken; G. W. Bruyn. (Amsterdam: North-Holland Publishing Co.); pp. 555-595; 1974.

A very comprehensive review is presented of features of papillomas of the choroid plexus, which can be divided into the rare villous hypertrophy, the benign papilloma forming the bulk of the case material, and the malignant papilloma and carcinoma, the true nature of some of which is in doubt. Discussion covers the prevalence with age, sex, and family history; pathology and histology; local effects of the papilloma; clinical features and course; diagnostic methods, including plain skull radiographs, pneumography, angiography, and gamma encephalography; and surgical treatment. Although infrequent in general neurosurgical practice, choroid plexus papillomas assume considerable importance in children, where they constitute one of the few types of intracranial tumors that are operable with some hope of permanent success. (186 references)

3671 EXPERIMENTAL BRAIN TUMOURS. (Eng.) Janisch, W.; Schreiber, D. *In: Handbook of Clinical Neurology. Tumours of the Brain and Skull: Part II.* Edited by P. J. Vinken; G. W. Bruyn. (Amsterdam: North-Holland Publishing Co.): vol. 17, pp. 1-41; 1974.

Studies of the induction and physiological consequences of brain tumors in experimental animals are reviewed. The main areas of discussion are: the simulation of space-filling intracranial or intraspinal lesions by mechanical means; the intracranial transplantation of malignant tumors; and the production of CNS tumors by viruses and chemical substances. (416 references)

3672 HEPATOCELLULAR CARCINOMA AND ERYTHROCYTOSIS. (Eng.) Davidson, C. S. (Clinical Res. Center, 50 Ames St., Cambridge, MA 02142). *Semin Hematol* 13(2): 115-119; 1976.

A small proportion of patients with primary liver cell cancer have erythrocytosis, which may develop to an extraordinary degree. A case history and a review of the reported incidences are presented. The disease occurs most often in males who present with cirrhosis and iron overload. Hypoglycemia and dysproteinemia may also be present. Several theories are discussed concerning the cause of the erythrocytosis, the most popular being an inappropriate production of erythropoietin by the tumor. This is supported by the observation that many of these patients have elevated plasma erythropoietin activity. Decreased degradation of the hormone may contribute to its increased activity, but the degree of this contribution has not been established. Deletion or degradation of an erythropoietin inhibitor may play a role in increasing its activity. The cause of erythrocytosis is hard to pinpoint since the disease occurs in several other apparently unrelated clinical situations, such as renal diseases, myomas of the uterus, and pheochromocytomas. (27 references)

3673 THYROID CANCER--IATROGENIC AND OTHERWISE. (Eng.) Stratton Hill, C., Jr. (Univ. Texas System Cancer Center, M.D. Anderson Hosp. and Tumor Inst., Houston, TX). *CA* 26(3): 160-164; 1976.

Differentiated thyroid cancer can be malignant or benign and can be induced by a moderate amount of radiation. Survival is measured in years for such patients, the treatment of choice being surgery for the primary disease or for recurrent and/or metastatic disease. Anaplastic thyroid carcinoma has a survival measured in months. The median survival from diagnosis to death is 3 mo, and most treatment results have been poor. A combination of radiation and actinomycin D has provided encouraging results in patients with tumors confined to the neck, but there is no effective treatment for recurrent or distant metastases. Medullary carcinoma of the thyroid is derived from a second epithelial cell system, which migrates from the neural crest into the thyroid during embryogenesis. This type occurs in both a sporadic and a familial form; fortunately, screening can be achieved by measuring serum calcitonin levels. A variety of other tumors may initially manifest themselves in the thyroid gland; they include squamous cell carcinomas, lymphomas, fibrosarcomas, hemangiosarcomas, osteogenic sarcomas, and chondrosarcomas. (4 references)

3674 CLONAL EVOLUTION AND PROGRESSION IN CHRONIC MYELOID LEUKAEMIA. (Eng.) Pedersen, B. (Inst. Cancer Res., Radiumstationen, DK-8000 Aarhus C, Denmark). *Blood Cells* 1(1): 227-237; 1975.

Literature concerning the clonal evolution of chronic myeloid leukemia (CML) is reviewed. In terms of physiology, kinetics, morphology, cytochemistry, and cytogenetics the granulopoietic cells of early CML appear to demonstrate no unequivocal signs of malignancy. After they are transformed into blastic crisis, however, subnormal proliferation and differentiation, cytochemical aberrations, and the appearance of aneuploid, Ph^1-positive hemopoietic clones indicate qualitative changes in their nature. Cytologically, the blastic crisis demonstrates features strikingly similar to those of acute myeloid leukemia. The kinetic, morphological, and cytochemical abnormalities are apparently secondary to the evolution of cytogenetically abnormal, granulopoietic clones; hence, the evaluation appears to occupy a central position in the natural history of the blastic crisis. (24 references)

3675 EWING'S SARCOMA. (Eng.) Nesbit, M. E. (Univ. Minnesota Medical Sch. and Mayo Memorial Hosp., Minneapolis, MN). *CA* 26(3): 174-180; 1976.

Ewing's sarcoma is a disease of children and young adults that may develop in any bone, but predominates in the shafts of the humerus, femur, and tibia. Asymptomatic and often clinically undedectable metastases are common. An extensive work-up is essential to diagnosis, which should include radiologic examination, a bone survey, isotopic scans, and open biopsy. The primary tumor can be controlled in over 60% of the patients with radiotherapy alone. However, cure is seldom effected due to dissemination to lungs, bone, and lymph nodes. One survey revealed a 5-yr survival rate of only 10% (36 of 374 children). Chemotherapy in addition to radiotherapy has been attempted by several investigators. Cyclophosphamide, vincristine, dactinomycin, and methotrexate have been used in various combinations, producing disease-free periods of 10-37 mo. A clinical trial using a standardized four-drug regimen and radiotherapy is underway that may extend the survival rates of these patients. (25 references)

3676 DIET AND CANCER OF THE COLON. (Eng.) Rowland, I. R. (No affiliation given). *Food Cosmet Toxicol* 14(3): 209-212; 1976.

In northwest Europe and the United States cancer of the large bowel is responsible for more deaths than any other type except lung cancer. The incidences of colon and breast cancer are very closely correlated and appear to have a common etiology. Positive correlations between cancer of the colon and cancer of the kidney, pancreas, and prostate have been found. All epidemiological studies have provided strong indications of the involvement of environmental factors in cancer of the colon. Additional supportive data have been gained for this theory from studies on immigrant populations moving from low to high risk areas. Mortality in these groups has risen to the level of the surrounding population. The incidence of cancer of the colon appears to reflect the degree of westernization of the diet. The causative agent is still unknown, but there is good evidence to implicate high dietary concentrations of fat and animal protein. Lack of dietary fiber also appears to be involved, probably because it decreases fecal bulk and thus increases the concentration of carcinogens in the gut. (45 references)

3677 SIGNIFICANCE OF CYTOCHEMICAL INVESTIGATIONS OF LEUKOCYTES IN CANCER DISEASE. (Rus.) Letskii, V. B. (N. N. Petrov All-Union Scientific Res. Inst. Oncology, USSR Ministry Public Health, USSR); Il'in, N. V. *Vopr Onkol* 22(2): 102-107; 1976.

Cytochemical methods help to elucidate and evaluate various metabolic characteristics of peripheral blood leukocytes in malignant neoplasms of different locations, and they can be employed successfully in diagnosis. Alkaline phosphatase activity, a widely used cytochemical parameter, is connected with phagocytosis and has also been shown to reflect nonspecific cellular defense mechanisms. Numerous investigations have revealed an increase in the alkaline phosphatase activity of neutrophils (APAN) in patients with cancer of the stomach, throat, mammary gland, lung, rectum, and uterine cervix, whereas this activity does not exceed the normal levels in benign tumors. A direct dependence has been observed between APAN elevation and the stage of malignancy. APAN changes significantly during surgical treatment of cancer patients, and an increase has been noted during localized thio-TEPA and dimethane therapy for lung cancer. In contrast, data on acid phosphatase activity in the blood cells of cancer patients are limited and contradictory. The cytochemical detection of glycogen, β-glucuronidase, and myeloperoxidase activity is also important for cancer diagnosis. (64 references)

3678 MULTIPLE PRIMARY NEOPLASMS. (Eng.) Schoenberg, B. S. *In: Persons at High Risk of Cancer. An Approach to Cancer Etiology and Control.* Edited by J. F. Fraumeni, Jr. (New York: Academic Press, Inc.): pp. 103-119; 1975.

Studies of multiple primary malignancies are reviewed and their implications for cancer control and etiology suggested. One study on nonsimultaneously diagnosed tumors, using a population-based central registry (Connecticut) indicated that individuals with one malignant neoplasm have 1.29 times the risk of developing a new independent primary tumor when compared to individuals who never had cancer. The increased risk of multiple primary tumors is highly selective on a site-specific basis. Site-group pairs with an excess of observed/expected second primary malignancies may be linked by common etiologic factors (environmental or genetic), by a previous treatment regimen, or by immune defects. The first category includes the development of a new colon malignancy following colon cancer, breast cancer following cancer in the opposite breast, lung cancer among bladder patients, and an excess of second primaries of the upper digestive and respiratory system in patients with scrotal carcinoma caused by mineral oil exposure. Mendelian inheritance has been described in several syndromes involving specific histologic types of multiple primary neoplasms. Familial polyposis of the colon may be associated with brain tumors in the Turcot syndrome or with osteomas, fibromas, lipomas, and epidermal cysts in Gardner's syndrome. Patients receiving radiation treatment for a first cancer of the uterine cervix or corpus are at increased risk for developing rectal carcinoma, and an excess of osteosarcomas, soft-tissue tumors, and skin cancers has been seen within the radiation field of children treated for retinoblastoma. Kidney transplant patients receiving immunosuppressive therapy have an increased risk of cancer, particularly lymphoreticular neoplasms and skin cancer. Patients with immunodeficiency syndromes have a high frequency of leukemia and lymphoreticular tumors. Studies of multiple primary malignancies are useful in detecting a second malignancy and help clarify the carcinogenic potential of various treatment modalities. (62 references)

3679 MORPHOGENETIC POTENTIAL OF TUMOR TISSUE. (Rus.) Golovin, D. I. (Leningrad, USSR). *Vopr Onkol* 21(11): 89-93; 1975.

The histology and etiology of mesotheliomas and myoepitheliomas, theories on the neurogenic origin of melanocytes and carcinoid tumors, the reversion of endocrine system tumors to a phylogenetically older form of tissue, and the relationship between the morphogenetic potential of mature tissue and dysembryogenesis are considered. (20 references)

3680 ONCOFETAL ANTIGENS AND METASTASIS MONITORING. (Eng.) Holyoke, E. D. *In: Fundamental Aspects of Metastasis.* Weiss, L., ed. (Amsterdam: North-Holland Publishing Co.): pp.407-423; 1976.

A review is presented of the discovery and current understanding of a number of oncofetal antigens: α-fetoprotein, carcinoembryonic antigen (CEA), α_2-H-fetoprotein, BS-fetoprotein, and fetal sulfoglycoprotein antigen. These antigens appear in fetal tissues or fluids and are reduced or absent in adults, but they reappear with malignancy. The use of CEA in particular as a diagnostic and prognostic monitor of colon cancer is discussed in detail. The extent of the disease is the most important determinant in the elevation of plasma or serum CEA in colon cancer, early lesions not being well-detected. Plasma CEA is generally felt to be elevated in less than 40% of Duke's A patients, 65% of Duke's B, 75% of Duke's C, and 90% of Duke's D patients. More-anaplastic tumors may shed CEA, resulting in elevated plasma levels but less abundant tissue levels. Initial CEA levels can be helpful in determining the prognosis in patients who undergo complete resection for cancer of the colon: only 8/23 patients who had no recurrence for an average of 14 mo had initial CEA levels greater than 2.5 ng/ml, and only 3 were greater than 4 ng/ml; conversely, 19/23 patients who developed recurrence had initial plasma values greater than 2.5 ng/ml and 14 were greater than 4 ng/ml. The association of CEA with cancers of the head, neck, lungs, breast, pancreas, stomach, genitourinary tract, and soft tissue is also discussed. (56 references)

3681 METASTASES: INCIDENCE, DETECTION, AND EVALUATION WITHOUT HISTOLOGIC CONFIRMATION. (Eng.) Gilbert, H. A.; Kagan, A. R. *In: Fundamental Aspects of Metastasis.* Weiss, L., ed. (Amsterdam: North-Holland Publishing Co.): pp.385-405; 1976.

Some guidelines are presented for the screening of major organs for metastasis in symptomatic and asymptomatic patients with cancer. The incidence of metastatic disease in certain key organs, both at diagnosis and at autopsy, has been listed for a large number of primary cancers. The incidences of primary breast cancer metastasis in bone, lung, liver, and brain were, respectively, 6%, 4%, 1%, and < 1% at diagnosis and 50%-85%, 60%, 45%-60%, and 15%-25% at autopsy. For many types of cancer, many metastases revealed at autopsy could not be detected at the time of diagnosis. Further discussion is made of diagnostic imaging studies, with particular reference to liver, bone, and brain scanning and lung-screening techniques. (82 references)

3682 OVERVIEW: HOST FACTORS. (Eng.) Miller, R. W. *In: Persons at High Risk of Cancer. An Approach to Cancer Etiology and Control.* Edited by J. F. Fraumeni, Jr. (New York: Academic Press, Inc.): pp. 121-128; 1975.

Host factors that predispose to cancer are reviewed. Wilm's tumor occurs in four growth excesses: malignant neoplasia, hamartomas, congenital hemihypertrophy, and the visceral cytomegaly syndrome. Groups at high risk of leukemia have in common a chromosomal abnormality seen in Down's syndrome, Fanconi's aplastic anemia, Bloom's syndrome, and chronic myelocytic leukemia. Chromosome abnormalities are also involved in certain forms of retinoblastoma in association with congenital malformations. Groups at high risk for lymphoma have in common an immunological abnormality. Environmentally induced immunosuppression (drugs given for renal transplantation) produces lymphoreticular neoplasia, sharing an effect in common with immunodeficiency diseases. Gonadoblastoma occurs only in patients with gonadal dysgenesis and only when a Y chromosome component is present in conjunction with the Turner phenotype. Pedigree analysis of familial cancers has indicated that the same combinations of cancers that occur excessively as second primaries also occur excessively among close relatives. In cancer-prone families, healthy members sometimes have the same laboratory aberrations found in the cancer-affected members, suggesting that these aberrations portend a high risk of cancer. In situ neuroblastoma (microscopic neuroblastoma) in infants under 3 mo who came to autopsy for noncancerous diseases was 40 times more common than clinical neuroblastoma. No in situ neuroblastoma was seen at autopsy in infants over 3 mo, indicating that the lesions regress or mature to normal tissue. Two infants with an unusual form of widely disseminated neuroblastoma are reported to have experienced total regression of the tumor without therapy by 1 yr. Identifying host factors that carry a high risk for cancer appears to be of greater value for studies of causation, which can lead to prevention, than for screening and early detection. (26 references)

3683 HEMOSTASIS AND CANCER. (Eng.) Brodsky, I.; Fuscaldo, A. A.; Fuscaldo, K. E. *In: Oncologic Medicine: Clinical Topics and Practical Management.* Sutnick, A. I.; Engstrom, P. F., eds. (Baltimore: University Park Press): pp.247-258; 1976.

The involvement of the blood coagulation system in neoplastic disease is discussed. Hemorrhage and/or thromboembolism are major causes of death in cancer patients: of 506 deaths in cancer patients, these complications were the main cause in 18% and a contributing cause in 43%. Neoplastic disease is a well-recognized cause of secondary thrombosis and the hypercoagulable state, presumably resulting from the release of thromboplastic substances from tumors. Fibrin deposition is important for the growth of primary tumors, the development of the tumor's own blood supply, and for aiding metastatic spread. Anticoagulants such as heparin and warfarin may affect metastasis by interfering with the lodgement of tumor cell emboli in capillary beds and, by further interfering with neovascularization, may also potentiate the action of chemotherapeutic drugs. Apparently, chemotherapy in leukemia can initiate disseminated intravascular coagulation, so that compensatory treatment with anticoagulants is required. L-Asparaginase treatment can cause hypofibrinogenemia by interfering with asparagine metabolism so that an abnormal fibrinogen with a decreased survival is produced. The destruction of leukemic cells in leukemic patients treated with brinase, a proteolytic enzyme that causes a sharp decrease in antiplasmin levels, suggests that the enzyme aided in the return of immunologic competence. The presumption is that the hypercoagulable state blocks the fibrinolytic mechanism necessary for the function of the immune mechanism. Virally altered megakaryocytopoiesis and thrombopoiesis could be the earliest manifestations of leukemia. Megakaryocytes and platelets in experimental animals are reservoirs and perhaps sites of replication of type C oncornaviruses, and electron microscopic studies have shown that viruslike particles with some of the characteristics of oncornaviruses are present in the platelets of patients with myeloproliferative disorders. Furthermore, reverse transcriptase, presumed of viral origin, has been detected in the platelets of patients with thrombocythemia and polycythemia vera. (27 references)

3684 ACQUIRED DISEASES. (Eng.) Templeton, A. C. *In: Persons at High Risk of Cancer. An Approach to Cancer Etiology and Control.* Edited by J. F. Fraumeni, Jr. (New York: Academic Press, Inc.): pp. 69-84; 1975.

The clinically apparent symptom-producing disease states that may result in an increased risk of cancer are reviewed. The incidence of cancer of the cervix is increased in patients with herpes genitalis because of herpes simplex virus type 2, but whether the relation is causal or due to some common third factor is not yet certain. Epstein-Barr virus has been associated with Burkitt's lymphoma and nasopharyngeal carcinoma and Australia hepatitus B antigen with hepatocellular carcinoma. Bacterial diseases predisposing to cancer are typically chronic infections with extensive fibrosis and persistent regenerative stress. It is difficult to separate the effects of bacterial products, scarring itself, regenerating cells, and treatment given. Schistosomiasis has been associated with bladder cancer in Egypt and the Gold Coast and *Clonorchis sinensis* infection is responsible for the high rates of intrahepatic bile duct cancer in the Far East. Gastritis and intestinal metaplasia are regularly found in association with stomach cancer. Patients with Crohn's disease have an elevated risk of adenocarcinoma of the small intestine, and 50%-90% of hepatocellular carcinoma occurs in association with cirrhosis. In surveys of cancer mortality in diabetes, a significant excess risk of pancreatic cancer has been seen consistently. Alterations in sex hormone production may result in clinically detectable diseases associated with cancer, and young women with the Stein-Leventhal syndrome are prone to various endometrial lesions, including well-differentiated adenocarcinomas. Theoretically, adaptation of cellular function by nutritional abnormalities, such as vitamin and protein deficiency or carbohydrate excess, might alter tumor incidence, but hard data are difficult to obtain. Chronic irritation is carcinogenic in some situations; for example, long-standing urinary stones may predispose to renal pelvic cancer, gallstones to cancer of the gallbladder, and thermal trauma to lip cancer in pipe smokers. Scars following burns, bites, infections, corrosive damage, and vaccinations have all been implicated. Operations such as tonsillectomy, splenectomy, appendectomy, and thymectomy may be risk factors, but there is no evidence of a significant hazard. (95 references)

3685 HEREDITARY FACTORS IN LEUKEMIA AND LYMPHOMA. (Eng.) Heath, C. W., Jr. *In: Cancer Genetics.* Edited by H. T. Lynch. (Springfield, IL: Charles C. Thomas): pp. 233-248; 1976.

The hereditary component of leukemia/lymphoma etiology is in all likelihood polygenic, its influence varying from one specific disease type to another. Although in no type has it been possible to distinguish hereditary influences from environmental ones, hereditary factors appear to play a lesser role in chronic granulocytic leukemia and Hodgkin's disease and a greater role in chronic lymphocytic leukemia (CLL). Distinct racial patterns exist in CCL (and possibly also in multiple myeloma); CCL also appears to have a greater tendency for cases to cluster in families than do other varieties of leukemia. Studies of leukemia incidence in twins have produced striking evidence of disease concordance in monozygous childhood twins that, however, may be less a matter of genetic origin than of antenatal leukemia spread by way of shared placental circulation. Chromosomal studies have not shown evidence of inherited cytogenetic defects that might be construed as genetic markers. However, genetic indicators of other sorts may be important: variations in immune capacity relative to leukemogenesis, variations in histocompatibility genotypes, and patterns of cellular response to viruses and other agents capable of triggering malignant disease. (159 references)

3686 FAMILIAL SUSCEPTIBILITY TO CANCER. (Eng.) Anderson, D. E. (Univ. Texas System Cancer Center, M. D. Anderson Hosp. and Tumor Inst., Houston, TX). *CA* 26(3): 143-149; 1976.

In families with a history of certain neoplasms, there is a greater risk for first-degree relatives than for the general population. Cancer of the stomach, breast, large intestine, uterus, and lung, childhood brain tumors, and sarcomas follow this pattern; relatives have a two- to fourfold increased risk. Genetic factors may also play a role in the etiology of leukemia occurring in siblings. Although heritable tumors exhibit a high degree of heterogeneity, they can be identified by the following characteristics: early age at onset or diagnosis, the tendency to develop at multiple sites in the same organ or bilaterally in paired organs, and limited range of phenotypic expression. A two-step model for childhood heritable tumors involves the first mutation being prezygotic and therefore present in all cells. Thereafter, only one event is required for tumor development. This model has been successfully applied to retinoblastoma and pheochromocytoma. (No references)

3687 FAMILIAL SUSCEPTIBILITY. (Eng.) Anderson, D. E. *In: Persons at High Risk of Cancer. An Approach to Cancer Etiology and Control.* Edited by J. F. Fraumeni, Jr. (New York: Academic Press, Inc.): 39-54; 1975.

Three methods used in genetic investigations of human cancer, retrospective studies, comparison of monozygotic and dizygotic twins, and pedigree studies, are reviewed and a two-step mutation model for carcinogenesis is described. The retrospective approach, which compares the morbidity and/or mortality rates in first-degree relatives of cancer patients with the rates in control relatives or with expected rates derived from population statistics, demonstrates a two- to fourfold excess of cancer of the stomach, breast, large intestine, uterus, and lung in relatives over controls. The increased risks are attributed to a genetic influence. Twin studies have indicated that the concordance rate in monozygotic twins is about double the rate in dizygotic twins for cancer of the stomach, breast, and intestine and for leukemia. Both studies have a number of methodological problems, specifically the etiological heterogeneity of cancers at a specific site. Evaluation of single families or pedigrees is perhaps the most meaningful approach for demonstrating a genetic basis for cancer susceptibility. A variety of neoplasms whose familial distribution patterns are consistent with Mendelian inheritance patterns have been identified. They are characterized by early average age at onset, development at multiple sites in the same organ or bilaterally in paired organs, and a limited range of phenotypic expression. These characteristics distinguish between genetic and environmental familial occurrences. The neoplasms with a dominant inheritance pattern agree with a two-step mutation model, in which the first mutation event is prezygotic and occurs in a germinal cell and the second is postzygotic. The carrier of this mutant gene may then develop zero, one, two, or more tumors in accordance with a Poisson distribution. The mechanisms through which susceptibility genes act and interact with environmental factors in carcinogenesis are largely unknown, but members of high-risk families should be counseled on the possible transmission of cancer susceptibility genes and the risks for subsequent tumor development. (62 references)

3688 CONGENITAL AND GENETIC DISEASES. (Eng.) Mulvihill, J. J. *In: Persons at High Risk of Cancer. An Approach to Cancer Etiology and Control.* Edited by J. F. Fraumeni, Jr. (New York: Academic Press, Inc.): pp. 3-37; 1975.

The cytogenetics of certain neoplasms, especially leukemia, and their meaning for cancer etiology, and

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the interrelationship of chromosomes, cancer, and congenital defects are reviewed. The Philadelphia chromosome (Ph^1) in chronic myelocytic leukemia and the absent chromosome 22 in meningioma are consistent chromosomal markers of neoplasia. Whether these abnormalities initiate malignancy or are just epiphenomena is unresolved, but tumor progression seems favored by increasing chromosomal aberrations. Banding techniques and computer technology have disclosed specific chromosomal markers in acute myelocytic leukemia, the Wilms' tumor-aniridia syndrome, and retinoblastoma in the 13q-syndrome. The coexistence of birth defects and neoplasia in Wilms' tumor suggests a common etiologic mechanism, perhaps in early gestation. The rate of leukemia in patients with Down's syndrome is at least 11 times normal; the risk of breast cancer in Klinefelter's syndrome approaches the risk in normal women but about 66 times the risk in normal men. The risk of gonadal malignancy in gonadal dysgenesis is about 25%. Of 72 cases tabulated, all had some cell lines with a Y chromosome, suggesting that the Y chromosome predisposes the dysgenetic gonad to malignancy. Of the known single-gene traits in man, 161 (9%) have neoplastic or preneoplastic manifestations or complications. Five Mendelian diseases that have chromosomal fragility (ataxia telangiectasia, Fanconi's anemia, Bloom's syndrome, Kostmann's infantile genetic agranulocytosis, and glutathione reductase deficiency) are of interest because they represent gross defects of DNA associated with malignancy and produced by a pair of mutant genes. Malignancies associated with hereditary disease affect every organ system and are often histologically identical to the most frequent human cancers (colon, breast, skin). More conditions are likely to be discovered as collaborative efforts recognize clinical, genetic, and laboratory manifestations of heterogeneity in existing diseases. (166 references)

3689 GENETIC DETERMINANTS OF CANCER. (Eng.)

Fraumeni, J. F., Jr. *In: Host Environmental Interactions in the Etiology of Cancer in Man. Proceedings of a meeting held at Primosten, Yugoslavia, 27 August-2 September 1972.* International Agency for Res. on Cancer (Lyon, France): pp. 49-55; 1973.

Patterns in the human cancer occurrence that indicate or suggest a genetic component in the origins of cancer are reviewed. A small proportion of human cancers fit Mendelian patterns of single-gene transmission and may be divided into two categories: (a) the mutant gene is expressed directly as a hereditary neoplasm (autosomal dominant), such as in retinoblastoma and intestinal polyposis; and (b) the gene results in a preneoplastic lesion or state that carries a higher risk of cancer, such as hamartomatous syndromes (autosomal dominant) and genodermatoses (autosomal recessive). Patients with genetic syndromes such as ataxia telangiectasia and the Wiskott-Aldrich syndrome, which feature defective immune systems, show a predisposition to lymphoreticular neoplasms. Common cancers show small familial risks that are derived empirically from statistical surveys; for adults the empirical risk among close relatives is about three-fold for carcinomas of the breast, stomach, colon, endometrium, prostate, and lung. Twin studies may help distinguish between environmental and hereditary factors; studies have shown 20% concordance in identical twins for childhood leukemia. Familial patterns from pedigree studies are consistent with the action of susceptibility genes. Pedigree studies have shown familial multiple cancer constellations that suggest neoplastic syndromes. Genetic factors or environmental differences or both may account for the racial and ethnic differences observed for certain cancers. relationships with certain blood groups or enzymes, such as the one discovered between stomach cancer and blood group A. Some nonfamilial malformations such as Down's syndrome may predispose toward cancer. Clinical, epidemiological, and laboratory indicators of genetic susceptibility should be useful in the early detection of cancer and for counseling to minimize added risks. (29 references)

3690 MOLECULAR GENETIC NATURE OF NEOPLASTIC TRANSFORMATION. (Rus.)

Shapot, V. S. (No affiliation given); Krechetova, G. D.; Likhtenshtein, A. V.; Shliankevich, M. A. *Patol Fiziol Eksp Ter* (2): 3-11; 1976.

Although the results of investigations have not excluded the possibility of mutation as a cause of neoplastic transformation, epigenomic changes are considered more probable. The literature suggests that local changes in the structure of chromosomes represent one of the mechanisms determining the distribution of active and silent genes. Genetic information is conveyed during transcription, during selective transfer of predecessors of informational RNA from the nucleus to the cytoplasm, and during realization of this information in the albumin-synthesizing apparatus; deviations characteristic of cancer cells can occur at each of these stages. Since the specific albumin, enzymes, and enzyme reactions accounting for the biologic properties of the neoplasm are absent, the polymorphism of biologically active substances may allow for the creation of new phenotypes in normal cell differentiation by different combinations of isofunctional compounds. (46 references)

3691 INTRODUCTION TO CANCER GENETICS. (Eng.)

Lynch, H. T. *In: Cancer Genetics.* Edited by H. T. Lynch. (Springfield IL: Charles C. Thomas): pp. 3-31; 1976.

A discussion is presented of the significance of genetic factors to the etiology of cancer. Consideration is given to the value of animal studies, including experiments with allophenic mice; the interplay between genetic and environmental factors; the importance of genetic factors in the heterogeneous human population; epidemiological studies; increased cancer incidence in particular pathological conditions; the segregation of genetic markers, such as glucose-6-phosphate dehydrogenase isozymes, within human tumors; family studies of childhood cancers; and the search for possible associations of increased cancer incidence with biological parameters such as the ABO blood group, the Australia antigen, and the HL-A histocompatibility antigens. In addition, the somatic mutation hypothesis and the suggestion that two mutations are required for cancer initiation, the first being genetically acquired by individuals who have a hereditary predisposition to cancer, are examined. As in experimental animals, the majority of tumors in man that appear to be under genetic control do not show simple inheritance; rather, they appear to be due to complex genetic mechanisms involving multiple genes in interaction with a variety of environmental factors. Certain animal strains can be so highly inbred as to manifest an exquisite genetic susceptibility to cancer; in this case it becomes difficult to evaluate the relative importance of nongenetic factors. On the other hand, various experimental studies involve such massive exposure to carcinogens that the role of host factors can be overwhelmed. (123 references)

3692 ENDOCRINE TUMOR GENETICS. (Eng.) Jackson, C. E.; *In: Cancer Genetics.* Edited by H. T. Lynch. (Springfield, IL: Charles C. Thomas): pp. 193-212; 1976.

A discussion is presented of the possible etiological significance of genetic factors to cancers of the pituitary, thyroid, parathyroid and adrenal glands, pancreas, and gonads. Heritable neoplasms may arise as a consequence of two mutational events, one mutation being inherited via germinal cells and the second occurring in somatic cells. The measurement of calcium-stimulated calcitonin levels in families of patients with medullary thyroid carcinoma-pheochromocytoma syndrome led to the discovery of three subjects with a previously undescribed condition of ''C-cell hyperplasia'' of the thyroid. This hyperplasia may represent the premalignant stage of the neoplasm and may be the consequence of inherited factors, occurring prior to a second stomatic mutation as required by the double mutation theory of hereditary cancer. (103 references)

3693 CYTOGENETICS WITH REFERENCE TO GONADOBLASTOMAS AND TO GONOCYTOMAS. (Eng.) Teter, J. *In: Cancer Genetics.* Edited by H. T. Lynch. (Springfield, IL: Charles C. Thomas): pp. 146-184; 1976.

A review is presented of the pathology and incidence of special gonadal tumors that occur in intersexual patients with a negative sex-chromatin pattern and of well-defined sex-chromosome anomalies in apparently phenotypic females with male karyotype 46,XY. The most characteristic tumor that occurs is the germ-cell tumor, which may be formed solely of gonocytes (gonocytoma-dysgerminoma) or mixed with smaller, dark or oval cells of the granulosa-Sertoli type and which may be classified as gonadoblastoma, dysgenetic gonadoma, or gonocytoma II or III. A conservative surgical approach (unilateral removal or bilateral adnexectomy) is recommended as the procedure of choice for gonocytoma (pure or mixed form) regardless of the patient's age and histological pattern. (104 references)

3694 ANENCEPHALUS, SPINA BIFIDA, TWINS, AND TERATOMA. (Eng.) Rogers, S. C. (4 Clifton Road, Rugby, England). *Br J Prev Soc Med* 30(1): 26-28; 1976.

A review of the literature on monoamniotic twin pregnancies indicated that congenital malformations may be the cause of the monoamniotic state. Twins with spina bifida and/or anencephalus are a rare occurrence; this malformation may be due to the breakdown of the interamniotic partition and a subsequent fetus-fetus interaction, leading to complete or partial destruction of one twin. Cells from the twin under attack may undergo somatic mutations, with loss of phenotypes, and then persist as teratoma or patches of aberrant skin with anomalous hair growth. The incidence of intraspinal and pineal teratoma may vary in time and place with spina bifida and anencephalus; the scalp type hair found over or around spina bifida lesions may, in male infants, carry female chromosomes. (22 references)

3695 CHROMOSOME CHANGES AND THE CLONAL EVOLUTION OF CANCER. (Eng.) Nowell, P. C. *In: Chromosomes and Cancer.* Edited by J. German. (New York, NY: John Wiley & Sons): pp. 267-285; 1974.

Tumor chromosomes were discussed in relation to the clonal aspect of neoplastic growth, with emphasis on the time when cytogenetically identifiable stemlines first appear during tumor development and on the nature and stability of the chromosome alterations as tumors progress. Most tumors, by the time they reach macroscopic size, have chromosome abnormalities that indicate a clonal pattern of neoplastic growth. Cytogenetically identifiable cell clones have been observed at all stages of tumor development. Chromosome abnormalities usually progress in parallel with other neoplastic characteristics, evolving toward more deviant karyotypes as the tumors become more malignant. These observations suggest that most neoplasms are unicellular in origin, that they develop through a microevolutionary process (with sequential selection of more neoplastic variants and more aneuploid karyotypes), and that the fully developed human malignancy is usually seen in an irreversible and individualized stage that may require individually designed therapy for systemic eradication. (31 references)

3696 CHROMOSOMES IN HUMAN CANCER. (Eng.) Rowley, J. D. (Dept. Medicine, Box 420, Univ. Chicago, 950 East 59th St., Chicago, IL 60637). *J Reprod Med* 17(1): 36-40; 1976.

Quinacrine fluorescence and Giemsa banding techniques are powerful tools for studying chromosomal aberrations in cancers, and they are used extensively to karyotype acute (AML) and chronic (CML) myelogenous leukemias. Patients with CML show a major chromosomal translocation. When their disease enters the terminal acute phase, additional abnormalities occur. The most common are additional C group chromosomes, specifically an extra No. 8. Patients with terminal phase AML also develop an extra No. 8 chromosome. Chromosome changes are also correlated to the degree of malignancy in reproductive tract carcinomas. The author presents the hypothesis that karyotypic change may be agent-specific and independent of the type of tumor produced. The study of chromosomal abnormalities may provide important clues to fundamental mechanisms of tumor progression in humans. (30 references)

3697 CURRENT EPIDEMIOLOGICAL EVIDENCE FOR TRANSMISSION OF HODGKIN'S DISEASE. (Eng.) Smith, P. G. (Dept. Epidemiology, Harvard Sch. Public Health, Boston, MA 02115); Pike, M. C. *Cancer Res* 36(2): 660-662; 1976.

The epidemiological evidence relating to the possibility of person-to-person transmission of Hodgkin's disease (HD) was examined. A situation was described in which 31 HD patients, diagnosed in the period 1950 to 1970 could be ''linked'' together either through direct personal acquaintance with each other or through mutual friendship with an unaffected person. These 31 patients represented 15% of the 208 HD patients diagnosed in Albany County during this time. Many studies proved difficult to interpret because of the absence of control data. It is concluded that, although certain studies are consistent with the hypothesis of person-to-person transmission, the evidence is at present weak and further epidemiological studies are needed. (22 references)

3698 AGE. (Eng.) Doll, R. *In: Host Environment Interactions in the Etiology of Cancer in Man. Proceedings of a Meeting Held at Primosten, Yugoslavia, 27 August-2 September 1972.* International Agency for Res. on Cancer (Lyon, France): pp. 39-48; 1973.

Four main patterns by which cancer incidence varies with age are described: one, typified by nephroblastoma, has an incidence peak in early life followed by a decline. Another, typified by bronchial carcinoma in men, shows a peak incidence in late middle life or early old age; a third, typified by stomach cancer in Europe, North America, and Australia, shows a rapid, uninterrupted regular increase from adolescence to old age; and the last, typified by breast cancer, is similar to the third except that after middle life the rate of increase slows down or ceases. Some cancers show a combination of patterns, such as acute leukemia; Hodgkin's disease has a pattern all its own. Failure to distinguish between histological and cytological types of cancer may account for the complex incidence patterns of some cancers; more than one type of disease may be lumped together. Studies concerning the four main age-incidence curves are reviewed. The many types of curves can be explained by the interaction between the duration and degree of exposure to a carcinogen, the activity of the exposed tissue, and its inherent susceptibility to cancer induction. There is no reason to suppose that the curve shape is dependent on any other ''aging'' process, whether in the tissue itself or in the animal as a whole. (16 references)

3699 GEOGRAPHICAL DIFFERENCES IN CANCER PATTERNS. (Eng.) Muir, C. S. *In: Host Environment Interactions in the Etiology of Cancer in Man. Proceedings of a Meeting Held at Primosten, Yugoslavia, 27 August-2 September 1972.* International Agency for Res. on Cancer (Lyon, France): pp. 1-13; 1973.

The major classes of geographical variation (international, within-country, and urban-rural) in cancer patterns are listed and discussed and their value in the creation and testing of causal hypotheses is examined. Concordance between cancer risk levels and the distribution of some habit or environmental factor do not guarantee any causal relationship; confirmatory studies are always necessary. The demonstration of a risk differential is not necessarily followed by a hypothesis for etiology. No general statement can arise from the conjunction of raw data; the contribution of investigative inspiration is noted. (68 references)

3700 EPIDEMIOLOGY AND ETIOLOGICAL STUDY OF HEMATOSARCOMAS IN CHILDREN. (Fre.) Munoz, N. (Centre International de Recherche sur le Cancer, O.M.S., 150, cours Albert-Thomas, 69008 Lyon, France); de-The, G. *C R Acad Sci [D] (Paris)* 282(13): 1329-1332; 1976.

Tumors are the second most frequent cause of death among children under 14 yr in developed countries. Inversely, in developing countries, they rank far below infectious diseases and nutritional deficiencies. Leukemia takes first place in highly industrialized countries with temperate climates. Its incidence is high, 9/100,000, with a peak between 0 and 4 yr. Boys seem to be stricken more often than girls. The level and age of maximal risk vary according to regions and ethnic groups. The lowest rate of occurrence is found among Black Americans and Africans; the highest, 11.4/100,000 in the 5-9 age group, belongs to American or European-born Jews residing in Israel. Lymphosarcomas (especially Burkitt's lymphoma in Equatorial Africa) have the lead in underdeveloped tropical countries. In these areas, it is not at all rare for boys from 5 to 9 to become victims of Hodgkin's disease, which is practically unknown in children of advanced nations. Epidemiology data lead to the formulation of etiological hypotheses. Childhood cancers may be linked to prenatal events. X-rays of the fetus increase the risk of leukemia. The influence of carcinogenic chemicals on intrauterine life has been established in five animal species. Some adenocarcinomas of the genital area that have appeared in teenage girls over the past decade seem to be due to diethylstilbestrol therapy received by their mothers during pregnancy. Congenital viral infections (German measles virus, herpes simplex virus, cytomegalovirus, some coxsackie viruses) are associated with birth defects. This teratogenic capacity may, or may not, be accompanied by a carcinogenic effect. Viral interventions have already been suspected in the development of some tumors (cancer of the uterine cervix and Hodgkin's disease). Burkitt's lymphoma remains the most familiar model of an association between a virus and a human tumor. (11 references)

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3736 DISCUSSION. PHOSPHOLIPID ASYMMETRY: STATUS AND PROSPECTS. (Eng.) Bretscher, M. S. *In: Cell Surfaces and Malignancy. John E. Fogarty International Center for Advanced Study in the Health Sciences and NIH.* Mora, P. T., ed. [Washington, DC; US Dept. Health, Education, and Welfare Publication No. (MIH) 75-796]: Fogarty International Center Proceedings No. 24, pp. 43-48; 1975. (10 references)

3737 VARIANTS OF HAMSTER FIBROBLASTS RESISTANT TO *RICINUS COMMUNIS* TOXIN (RICIN). (Eng.) Meager, A. (Natl. Inst. for Medical Res., Mill Hill, London NW7 1AA, U.K.); Ungkitchanukit, A.; Hughes, R. C. *Biochem J* 154(1): 113-124; 1976.

Variant baby-hamster kidney (BHK) cell lines were isolated that grew in the presence of high concentrations of ricin, the toxic lectin of castor beans (*Ricinus communis*). The variant lines were independently derived from several cultures of normal BHK cells that had been exposed to the mutagen N-methyl-N-nitro-N-nitrosoguanidine before selection by ricin. The cell lines maintained a high degree of resistance to recin after growth in lectin-free medium for prolonged periods and therefore exhibited stable phenotypes that were different from normal BHK cells. A preliminary classification of the phenotypes was made. Several cell lines bound normal amounts of ^{125}I-labeled ricin, whereas others bound the lectin poorly. A loss of surface receptors for two other lectins, *R. communis* RCA$_1$ and *Axinella polyploides*, which had specificities similar to ricin, was also found in some but not all of the cell lines showing decreases surface concentrations of ricin receptors. The binding to the recin-resistant cells of lectins of different sugar specificity (*Lens culinaris* lectin and concanavalin A) was similar to, or higher than, normal BHK cells. Several of the recin-resistant cell lines were shown to be cross-resistant to the weak cytotoxicity of *Phaseolus vulgaris* lectin.

3738 CALCIUM-DEPENDENT STIMULATION OF BALB/C 3T3 MOUSE CELL DNA SYNTHESIS BY A TUMOR-PROMOTING PHORBOL ESTER (PMA). (Eng.) Boynton, A. L. (Div. Biological Sciences, Natl. Res. Council Canada, Ottawa, Canada K1AOR6); Whitfield, J. F.; Isaacs, R. J. *J Cell Physiol* 87(1): 25-32; 1976.

During their first six passages, BALB/c 3T3 mouse cells did not proliferate in serum containing-medium having an ionic calcium concentration of 0.05 mM or less. By the ninth passage, however, they were able to multiply in the presence of these lower calcium levels. In low calcium (0.02 mM) medium, passage 1-6, cells in sparse cultures were blocked at the G1/S boundary of their cycle. These blocked cells could be induced to start making DNA within only 1 hr either by returning the ionic calcium level to a normal range of values (1.25 mM), or by adding 0.05 μg/ml of PMA (12-O-tetradecanoylphorbol-13-acetate). PMA probably acted by sensitizing the blocked cells to calcium rather than replacing the ion, since it was ineffective in ionic calcium-free medium. PMA did not by itself induce proliferation of cells (regardless of the number of passages) that had been proliferatively inactivated by density-dependent factors in confluent cultures. PMA did promote DNA synthesis by these cells during their brief transition to the ''cycling'' state caused by exposure to fresh serum-containing medium.

3739 MORPHOLOGICAL CHANGES IN RAT BRAIN AND SKELETAL MUSCLE IN THE REGION OF NICKEL OXIDE IMPLANTATION. (Eng.) Sosinski, E. (Medical Acad., Poznan, Poland). [illegible]atol Pol 13(3/4): 479-483; 1975.

To investigate the carc[illegible] effects of powdered nickel oxide (Ni_2O_3), 3-[illegible]es were administered subcortically to the right f[illegible]tal area of 10 white Wistar rats, and 10 mg powdered Ni_2O_3 was implanted im into the left gastrocnemius. The intracerebrally administered Ni_2O_3 induced neoplastic growth in the CNS; these neoplasms were of the sarcoma or meningioma type. The tumors presumably originated from connective tissue and meningoendothelial cells; there was no glial tissue reaction. In the skeletal muscle implants, a gradually increasing reaction of connective tissue, phagocytic cells, and plasma cells was noted.

3740 INDUCTION OF FIBROSARCOMA BY ADMINISTRATION OF DMBA INTO MANDIBULAR SOCKETS OF RATS. (Eng.) Sela, J. (Hadassah Medical Sch., P.O. Box 1172, Jerusalem, Israel). *Experientia* 32(4): 516-517; 1976.

Ten albino Hebrew University (Sabra) strain rats were injected with pellets of 0.2 ml Vitepsol H-15 containing 10% 9,10-dimethyl-1,2-benzanthracene into the site where the first molar had been extracted. When they were sacrificed 9 mo later, it was found that 6 of the 10 had developed fibrosarcomas (4-6 cm) and that the neoplasms had invaded the adjacent tissue. Two rats died before the experiment was completed and another two, along with 20 controls, failed to develop any neoplastic growth.

3741 ADDUCT OF TYROSINE AND THE ONCOGEN 3-ACETOXYXANTHINE. (Eng.) Stohrer, G. (Memorial Sloan-Kettering Cancer Center, New York, NY 10021); Brown, G. B. *Biochemistry (Washington)* 15(13): 2772-2775; 1976.

Two products are describ[illegible]n the reaction of L-tyrosine (I) (500 m[illegible] mmol) with the activated oncogen 3-acetoxy[illegible]ine (II) (1.0 g, 3.7 mmol) at pH 8.0. A major product of the reaction was characterized as 8-xanthin[illegible]-4'-O-tyrosine (III). A similar product was forme[illegible]h tyrosylglycine. Another major product was [illegible] hydantoin of tyrosine. III was hydrolyzed in acid [illegible] yield tyrosine and uric acid, but was stable [illegible] base. The synthesis of 8-phenoxyxanthine is also described. A metabolite identical with III in several chromatographic systems was found in the urine of two male Sprague-Dawley rats after ip administration of radioactive 3-hydroxyxanthine. This r[illegible]ented about 0.05% of the urinary radioactivit[illegible] the purine N-oxides, all oncogenic esters rea[illegible] tyrosine, while nononcogenic esters do [illegible] the products formed with 3-acetoxyxanthine [illegible]ified, but the chromatographic propert[illegible]ducts with other purine esters suggest s[illegible]uctures. (16 references)

3742 NUCLEOPHILIC DISPLACEMENT ON THE ARENE OXIDES OF PHENANTHRENE. (Eng.) Bruice, P. Y. (Dept. Chemistry, Univ. California, Santa Barbara, CA 93106); Bruice, T. C.; Yagi, H.; Jerina, D. M. *J Am Chem Soc* 98(10): 2973-2981; 1976.

The reactivity of nitrogen, oxygen, and sulfur nucleophiles on phenanthrene 9,10-oxide (the K region arene oxide of phenanthrene) and, for comparison, on phenanthrene 1,2-oxide and phenanthrene 3,4-oxide (the isomeric non-K region arene oxides) was studied. Phenanthrene 9,10-oxide underwent nucleophilic attack by oxygen bases and a wide variety of amines as a result of its relatively slow rate of ring opening to carbonium ion and decreased rearrangement rate of the carbonium ion to phenol. The reaction rate of this oxide with nucleophiles was comparable to that of ethylene oxide. The second-order rate constants for attack by primary and secondary amines gave a β nuc value of 0.4. Certain tertiary amines (trimethylamine and quinuclidine amines) exhibited enhanced nucleophilic reactivity. This may be due to preequilibrium complex formation prior to nucleophilic attack. The quinuclidine amines gave a βnuc value of 0.1. Oxygen bases were less reactive nucleophiles and gave a βnuc value of 0.2. With the non-K region arene oxides (phenanthrene 1,2-oxide, phenanthrene 3,4-oxide, benzene oxide, and naphthalene oxide), nucleophilic attack by amines and oxygen bases was not sufficiently rapid to compete with the spontaneous aromatization reaction. Thiolate anions exhibited considerably greater nucleophilic reactivity than amines and, consequently, reacted with both the K region and non-K region arene oxides. The second-order rate constants for attack by thiolate anions resulted in a βnuc value of 0.2 for each of the arene oxides. Glutathione was no more reactive a thiol than would be predicted from its p*K*a. NMR studies of the nucleophilic adducts showed that nucleophilic attack was stereospecific and resulted in trans adducts. Thus either direct SN2 attack or nucleophilic trapping of tight ion pairs occurs. An index of nucleophilic susceptibility was defined for epoxides and arene oxides: ethylene oxide = 1, phenanthrene 9,10-oxide = 0.3, and the non-K region arene oxides are all < 0.01.

3743 EVIDENCE FOR CAFFEINE-SENSITIVE DAMAGE IN METHYLAZOXYMETHANOL ACETATE-TREATED L5178Y CELLS.

(Eng.) Shinohara, K. (Div. Biology, Natl. Inst. Radiological Sciences, Anagawa, Chiba-shi, Chiba 280, Japan); Matsudaira, H. *Chem Biol Interact* 12(1): 53-63; 1976.

The effect of methylazoxymethanol acetate (MAMA) on colony survival, cell proliferation, and DNA synthesis of murine lymphoma L5178Y cells were studied. Decreased colony survival and immediate depression of cell proliferation and DNA synthesis was found in L5178Y cells treated with 500 or 250 μg/ml MAMA for 1 hr at 37 C. Prelabeling with 5-bromodeoxyuridine did not enhance significantly the carcinogen-induced cell lethality. Posttreatment with 0.4-0.6 mM caffeine greatly enhanced cell lethality and depression of cell proliferation. These effects of caffeine were diminished when the cells had passed through two generations following the MAMA treatment. Experiments involving MAMA treatment at 2.5 hr after the release of synchronized cells from a colcemid block showed that the action of caffeine was located primarily in S phase following the MAMA treatment. These results suggest that in L5178Y cells, MAMA induces DNA damage; it is concluded that this damage is repaired by a mechanism analogous to postreplication repair of UV light-induced damage.

3744 A THERMOSTABLE ANTIGEN CHARACTERISTIC FOR CARCINOGEN-INDUCED RAT INTESTINAL TUMORS.

(Eng.) Abeyounis, C. J. (Sch. Medicine, State Univ. New York at Buffalo, Buffalo, NY 14214); Milgrom, F. *J Immunol* 116(1): 30-34; 1976.

The physicochemical and serologic properties of carcinogen-induced intestinal tumors of the rat and their relationship to human carcinoembryonic antigen (CEA) were studied. Precipitation tests using properly absorbed rabbit antisera revealed that perchloric acid extracts of 1,2-dimethylhydrazine-induced jejunal and colonic adenocarcinomas from Fischer-344 rat tissue contained an antigen not detectable in extracts of normal Fischer rat tissue or other rat tumors (methylcholanthrene-induced tumors, Wilms' tumor, and polyoma-induced tumor). This rat tumor antigen was detected in extracts of normal rat colon by employing the much more sensitive procedure of tanned cell hemagglutination inhibition; however, the concentration of the antigen in tumor was about 250-fold higher than in normal colon. Similar to human CEA, the rat tumor antigen was found to be heat stable, contained glycoprotein, migrated as a β-globulin, and appeared at a high concentration in fetal intestines; however, the rat tumor antigen failed to produce a precipitation reaction with human CEA preparations.

3745 THE METABOLISM OF L-ETHIONINE IN RATS FED ALPHA-NAPHTHYL ISOTHIOCYANATE.

(Eng.) Brada, Z. (Papanicolaou Cancer Res. Inst., Miami, FL 33136); Bulba, S. *Res Commun Chem Pathol Pharmacol* 13(1): 19-26; 1976.

A study was conducted on the metabolism of L-ethionine in CFN rats fed α-naphthyl isothiocynate in order to correlate changes of ethionine metabolism with morphological alterations in the liver. L-[Ethyl-1-^{14}C]ethionine was administered in a single dose by gavage to rats fed a diet containing 0.10% α-naphthyl isothiocyanate for 99 days. The analysis of ethionine and its metabolites in the urine was performed chromatographically on an AG 50W-L12 column. Ethionine metabolites excreted into urine were investigated as a function of induced pathologic changes in the liver. A short-time exposure (6 and 14 days) to α-naphthyl isothiocyanate increased the excretion of free ethionine and S-adenosylethionine at the expense of total ethionine sulfoxide. With the extended feeding period the excretion of total ethionine sulfoxide became normalized, while the excretion of free ethionine and S-adenosylethionine decreased. The acetylated fraction of ethionine sulfoxide decreased rapidly and reached a minimum in 14 days of feeding. This fraction subsequently started to increase, but did not reach the values obtained in normal rats. This comparison of the ethionine metabolism and the histopathological picture of liver does not demonstrate any direct relationship between the changes in the ethionine metabolism and the replacement of the liver parenchyma proliferating ductular cells and fibroblasts, which occurs following treatment with α-naphthyl isothiocyanate.

3746 ON THE FORMATION OF N-NITROSODIETHANOLAMINE FROM A GRINDING FLUID UNDER SIMULATED GASTRIC CONDITIONS. (Eng.) Zingmark, P.-A. (Dept. Organic Chemistry, Umea Univ., S-901 87 Umea, Sweden); Rappe, C. *Ambio* 5(2): 80-81; 1976.

Since N-nitrosamines cause cancer in rats and are formed in vivo in the gastric juices in man, experiments were conducted to measure N-nitrosodiethanolamine concentrations in the grinding fluid of machines. The fluid, which consists of triethanolamine, sodium nitrite, water, and fluorescein, was kept at 37 C for 1 hr in buffer solutions at different pH values. At pH 2, the yield per 1 ml of grinding fluid was 13 mg N-nitrosodiethanolamine, 1,000 times the level found in certain foodstuffs, and at pH 3.9 it was 22 mg. Therefore, precautions should be taken so that workers around a machine with mist containing N-nitrosamines do not inhale any of the mist.

3747 CYTOTOXICITY OF HAIR COLOURANT CONSTITUENTS: CHROMOSOME DAMAGE INDUCED BY TWO NITROPHENYLENEDIAMINES IN CULTURED CHINESE HAMSTER CELLS. (Eng.) Kirkland, D. J. (Royal Cancer Hosp., Pollards Wood Res. Station, Nightingales Lane, Chalfont St. Giles, Bucks., U.K.); Venitt, S. *Mutat Res* 40(1): 47-55; 1976.

Two aromatic amines 2-nitro-p-phenylenediamine (2NPPD) and 4-nitro-o-phenylenediamine (4NOPD) are constituent dyes of widely used proprietary hair colorants. Both compounds were cytotoxic (in the dose-range 5-100 μg/ml) to CHMP/E cells (a recently-derived near-diploid clone of Chinese hamster prostate cells) following 5 days continuous exposure. 2NPPD was more toxic than 4NOPD at equivalent doses. These cytotoxic effects were paralleled by a time-dependent increase in the yield of chromosome aberrations following exposure of CHMP/E cells to 25 μg/ml 2NPPD or 4NOPD. The increase in the incidence of breaks and exchanges was particularly marked after 4 and 7 days exposure; 2NPPD caused more chromosome damage than 4NOPD at equivalent doses. The use of 2NPPD and 4NOPD as hair-colorant constituents might be associated with a long-term health hazard. Attention was drawn to established correlations between these biological effects and carcinogenicity and to epidemiological studies suggesting that the elevated incidence of certain tumors in hairdressers and in those engaged in cosmetics manufacture might be due to exposure to chemical carcinogens.

3748 THIN-LAYER CHROMATOGRAPHY OF N-NITROSAMINES. (Eng.) Gunatilaka, A. A. L. (Chemistry Dept., Univ. Sri Lanka Peradeniya Campus, Peradeniya, Sri Lanka). *J Chromatogr* 120(1): 229-233; 1976.

A homologous series of alkylmethylnitrosamines was subjected to thin layer chromatography with a silica gel G support; developed by solvents containing various proportions of n-hexane, diethyl ether, and chloromethane; and stained with an acidic iodoplatinate reagent. The Rf values indicated that the functional group N-NO is a major factor in determining the degree of absorption onto the silica gel. (15 references)

3749 EFFECT OF PHENOBARBITAL STIMULATION ON MORPHOLOGICAL AND CYTOENZYMATIC CHANGES IN THE LIVER OF BENZENE INTOXICATED RATS. (Ger.) Jonek, J. (Biologisch-Morphologisches Institut, Schlesische Medizinische Akademie Katowice, Katowice, Poland); Kaminski, M.; Grzybek, H.; Panz, B.; Gruszeczka, B. *Acta Histochem (Jena)* 55(1): 60-79; 1976.

Smooth endoplasmic reticulum (ER) proliferation as an adaptation to increased catabolism or as a direct result of cell injury in benzene intoxication was examined. Whether phenobarbital-induced proliferation of smooth ER in hepatocytes in benzene toxicity can elicit an elevated metabolic breakdown of benzene is also discussed. Two groups of 20 male Wistar rats were used. The controls received 60 mg/kg phenobarbital ip once daily for 3 days. The experimental group was similarly prepared, but also received 1,000 mg/kg benzene ip on day 3 shortly after the last phenobarbital injection. Animals were sacrificed in 24 hr. The control hepatocytes showed both regularly oriented light and irregularly shaped dark cells, depending on the electron density, with ring-shaped structures. In experimental hepatocytes, there were more dark cells, but the light cells also contained ring-shaped structures. A well-developed rough ER occurred in both cell types. Dark cells were localized chiefly in the central lobular regions of relatively low metabolic activity. A paucity of organelles, low levels of glucose-6-phosphatase and $NADPH^2$-tetrazole reductase, a well-developed smooth ER, and low mitochondria and lysosome contents were typical of experimental dark cells. The experimental light cells exhibited a weak smooth ER, high glucose-6-phosphatase, and high nucleoside diphosphatase. Light cell behavior is probably linked to benzene biotransformation, while the dichotomy of the light and dark cells requires both benzene and phenobarbital. (18 references)

3750 HEMIN REVERSAL OF BENZENE-INDUCED INHIBITION OF RETICULOCYTE PROTEIN SYNTHESIS. (Eng.) Forte, F. J. (New York Univ. Sch. Medicine, 550 First Ave., New York, NY 10016); Cohen, H. S.; Rosman, J.; Freedman*, M. L. *Blood* 47(1): 145-154; 1976.

Studies were undertaken to investigate if benzene inhibits translation in reticulocytes at the hemin-dependent site. Reticulocyte-rich blood was collected in heparin by cardiac puncture of phenylhydrazine-treated rabbits. Human reticulocytes were obtained from two patients with sickle cell anemia. Protein synthesis was determined by measuring L-(U)-^{14}C-leucine incorporation, and heme synthesis in intact cells was determined by the incorporation of L-2-^{14}C-glycine into hemin. Benzene (0.056-0.113 M) rapidly and reversibly inhibited protein synthesis in anucleate human sickle cell and rabbit reticulocytes. Hemin (50 μM) both prevented and reversed this effect of benzene. The inhibition in rabbit reticulocytes was accompanied by a conversion of polyribosomes to monoribosomes. The polyribosomal disaggregation required ribosomal movement along messenger RNA and was also prevented and reversed by 50 μM hemin. Benzene was also shown to inhibit heme synthesis in rabbit reticulocytes. ATP concentration, as determined in perchloric acid extracts of reticulocytes, was unaffected by benzene. In addition, colorimetric analysis of glutathione levels in the reticulocytes revealed no alteration. A translational repressor (HCR) of reticulocyte cell-free protein synthesis was isolated from intact cells incubated with benzene, while no significant amount of HCR was found in cells incubated with both benzene and hemin. These results indicate that benzene inhibits translation at the heme-dependent site of initiation. The clinical implications of these experiments remain to be elucidated.

3751 HEPATIC AND EXTRAHEPATIC METABOLISM, IN VITRO, OF AN EPOXIDE (8-^{14}C-STYRENE OXIDE) IN THE RABBIT. (Eng.) Jamis, M. O. (Pharmacology Branch, Natl. Inst. Environmental Health Sciences, Natl. Insts. Health, Research Triangle Park, NC 27709); Fouts, J. R. ; Bend, J. R. *Biochem Pharmacol* 25(2): 187-193; 1976.

The suggestion that epoxide metabolites may be mediators in the carcinogenicity of aromatic hydrocarbons was investigated. Two epoxide-metabolizing enzymes, glutathione-S-epoxide transferase and epoxide hydrase, were studied in subcellular fractions of rabbit liver, lungs, intestinal mucosa and kidney. Glutathione-S-epoxide transferase in soluble fraction was assayed by a new specific radiochemical method using styrene oxide (-8-^{14}C) as substrate. Liver had the highest specific activity of each enzyme, but there was no correlation between the relative specific activities of glutathione-S-epoxide transferase and epoxide hydrase in the extrahepatic organs. Feeding rabbits a purified diet did not alter the specific activities of epoxide hydrase or glutathione-S-epoxide transferase in liver and intestine. Rat and guinea pig had much higher specific activities of glutathione-S-epoxide transferase than rabbit in liver and kidney, and slightly higher specific activities in lung and intestine. Species did not differ markedly in epoxide hydrase activities when the same organ (liver, lung, intestine or kidney) was compared in rabbits, rats and guinea pigs, except that rat intestine had much lower epoxide hydrase activity than intestine from rabbit or guinea pig.

3752 BIOCHEMICAL PARAMETERS OF BHT-INDUCED CELL GROWTH IN MOUSE LUNG. (Eng.) Witschi, H. (Departement de Pharmacologie, Faculte de Medecine, Universite de Montreal, Montreal, Quebec, Canada); Kacew, S.; Tsang, B. K.; Williamson, D. *Chem Biol. Interact* 12(1): 29-40; 1976.

Experiments were conducted to determine if the prolifertive response of lung cells to butylated hydroxytoluene (BHT) previously found in some mice could be elicited in various mouse strains and whether it was possible to detect and measure cell growth in lung with a convenient biochemical parameter. Male mice of 7 different strains (C57, Swiss/Webster, DBA2, C3H, BALB/c, ARS Sprague-Dawley, BDF$_1$, and C57 x C3H) were injected ip with 400 mg/kg of BHT. Two and 4 days later, the incorporation of thymidine into pulmonary DNA was significantly increased in all treated animals, and this was accompanied by an increase in lung wt and pulmonary DNA. Thymidine kinase activity and DNA polymerase activity were enhanced in the lungs of BHT-treated animals, and maximum activity of these enzymes appeared to precede maximum thymidine incorporation by 23 hr. Three days after BHT treatment, a good correlation was found between administered dose and thymidine kinase activity. Measuring the activity of this enzyme might serve as a convenient biochemical marker to follow and to quantitate BHT-produced cell proliferation in lung. The concentrations of cyclic AMP and the activity of adenylate cyclase were not altered by BHT on days 1-9 after administration. BHT produced also some dose-dependent, time-dependent increases in the activities of pulmonary 5'-nucleotidase and glucose-6-phosphate dehydrogenase but had little effect on isocitric dehydrogenase, pyruvate kinase, and lactic dehydrogenase.

3753 BEHAVIOR AND FATE OF ETHYLENETHIOUREA IN PLANTS. (Eng.) Hoagland, R. E. (Southern Weed Science Lab., Agricultural Res. Service, U.S. Dept. Agriculture, Stoneville, MS 38776); Frear, D. S. *J Agric Food Chem* 24(1): 129-133; 1976.

The uptake, movement, persistence, and degradation and/or metabolism of [4,5-^{14}C]ethylenethiourea (ETU) were studied in several mono- and dicotyledonous plants. When young seedlings and excised leaves of corn, lettuce, pepper, and tomato were pulse-treated with aqueous ETU (1.77 μg, 2-4 hr), the labeled ETU was readily absorbed by either the roots or the petioles of excised leaves and was translocated primarily via the xylem. After 20 days, only 1-2% of the initial dose remained as [^{14}C]ETU, but several degradation and/or metabolic products were present in the methanol-soluble extracts, and part of the ^{14}C was found in the methanol-insoluble residue. Methanol-soluble and -insoluble degradation products were determined in the various tissue sections for a period of 20 days after treatment. Only minor amounts of $^{14}CO_2$ were obtained from treated plants or excised leaves, but a major degradation product was isolated from pepper plants and was tentatively identified by infrared and mass spectroscopy as ethyleneurea (EU), the oxygen analog of ETU. The results demonstrate that ETU is readily absorbed and translocated by these plants. Complete ETU degradation to CO_2 is a slow and minor catabolic pathway in higher plants.

3754 EMBRYONAL NEOPLASMS IN THE OPOSSUM: A NEW MODEL FOR SOLID TUMORS OF INFANCY AND CHILDHOOD. (Eng.) Jurgelski, W., Jr. (National Inst. Environmental Health Sciences, Res. Triangle Park, NC 27709); Hudson, P. M.; Falk, H. L.; Kotin, P. *Science* 193(4250): 328-332; 1976.

The postnatal completion of embryonic and fetal development in the opossum offers advantages over typical eutherian species for experimental oncogenesis, especially embryonic tumor induction. A series of 532 opossums was given 100 mg/kg of ethyl nitrosourea (ENU) po, either as a single dose or in four increments of 25 mg/kg every other day. The animals ranged in age from under 1 day to 16 wk. A variety of mesenchymal and epithelial neoplasms, including embryonic neoplasms of the eye, liver, brain, kidney, muscle, and jaw, developed in the opossums treated with ENU. Three of the embryonic tumors (teratoid medulloepithelioma, ganglioglioma, and myxoma) have not been previously induced by systemic administration of a carcinogen. Unilateral, malignant, neuroectodermal intraocular tumors developed in five animals exposed to ENU between 7 and 21 days of age. Embryonal tumors of the kidney were found in 30% of animals exposed to ENU 6 wk after birth. Several forms of neurogenic neoplasms were identified in opossums exposed to ENU within 8 wk of birth. Several different types of mesenchymal tumors of soft tissue were found in opossums given ENU at various stages of postnatal development. Both epithelial and mesenchymal embryonal tumors were induced in the jaws of opossums given ENU through 12 wk of age. Several types of hamartomas and malformations developed in opossums given ENU 3 wk after birth.

3755 IN VITRO TRANSFORMATION: INTERACTIONS OF CHEMICAL CARCINOGENS AND RADIATION. (Eng.)

DiPaolo, J. A. *In: Biology of Radiation Carcinogenesis.* Edited by J. M. Yuhas, R. W. Tennant, and J. D. Regan. (New York: Raven Press): pp. 335-342; 1976.

To determine the factors that influence transformation and the degree to which any agent acts alone or with another agent (from the same or a different category of carcinogen), the interaction of potent chemical carcinogens in combination with either weak chemical carcinogens (methylmethane sulfonate) or physical agents (UV and x-rays) was studied. The use of benzo(*a*)pyrene (BP) on Syrian hamster cell strains resulted in a dose-response relation consistent with a Poisson distribution; this indicates that the transformation phenomenon is inductive. The joint action or interaction of BP with other agents occasionally resulted in an increased incidence of transformation. The pretreatment of the cells with ionizing radiation (250 R) or alkylating chemicals enhanced the frequency of transformation on a cell or colony basis ordinarily obtained with known chemical carcinogens. Pretreatment with nonionizing irradiation (UV, 254 nm) did not have a similar effect. The two types of irradiation and the alkylating agents reduced the cloning efficiency of the cells. X-ray alone produced no transformation; the alkylating chemicals alone produced transformations infrequently, whereas UV alone also produced a significant number of transformations. The number of transformations associated with UV was increased by pretreating the cells with x irradiation. Transformation enhancement by x ray or x ray-type agents appears to be independent of the type of second carcinogen used.

3756 MODIFICATION OF INTERSTITIAL FLUID CLOSE TO CHEMICALLY-INDUCED TUMOURS AND CORRELATION WITH A GROWTH STIMULATION OF TARGET CELLS.

(Eng.) Vaillier, D. (Res. Unit Experimental Cancerology Radiobiology, U95-INSERM, 54500 Vandoeuvre-lez-Nancy, France); Vaillier, J.; Donner, M. *Eur J Cancer* 12(2): 125-129; 1976.

Acrylamide gel electrophoresis was used to investigate the composition of the sc interstitial fluid in C3H/He, Swiss/B, and CBA mice bearing methylcholanthrene- and benzpyrene-induced transplantable rhabdomyosarcomas. Diffusion chambers containing cells from methylcholanthrene-induced tumors (MC2 or MC3) were implanted sc into normal and tumor-bearing mice. The sc interstitial fluid proteins were analyzed by electrophoresis in 7.5% polyacrylamide gels, and the total protein content was determined according to Lowry's method. The growth of MC3 target cells in the diffusion chambers was significantly increased in the MC3-bearing mice compared with the non-tumor-bearing controls. A similar result was not obtained with the MC2 tumor. It appeared that the MC3 tumors influenced the growth stimulation of MC3 target cells through the sc interstitial fluid. Some of the protein fractions were significantly decreased in the sc interstitial fluid of the stimulated tumor-bearing mice. The serum and peritoneal fluid showed no such changes. The results suggest that some tumors may use growth inhibitory substances that occur naturally in sc interstitial fluid.

3757 IDENTIFICATION OF MUTAGENIC METABOLITES OF BENZO[a]PYRENE IN MAMMALIAN CELLS. (Eng.)

Huberman, E. (Dept. Genetics, Weizmann Inst. Science, Rehovot, Israel); Sachs, L.; Yang, S. K.; Gelboin, H. V. *Proc Natl Acad Sci USA* 73(2): 607-611; 1976.

The mutagenicity of benzo[a]pyrene and 15 of its derivatives (phenols, the benzo[a]pyrene-1,5-epoxide, dihydrodiols, two isomeric 7,8-diol-9,10-epoxides, a 6-methyl derivative, and a 6-hydroxymethyl derivative) were tested with Chinese hamster V79 cells. Mutations were characterized by resistance to ouabain or S-azaguanine. Mutagenesis was tested both in the presence and absence of benzo[a]pyrene-metabolizing normal golden hamster cells. All the tested phenols, 4,5-diols, trans-9,10-diol, 6-methyl, and 6-hydroxymethyl derivatives of benzo[a]pyrene showed little or no mutagenicity for both genetic markers. The (±)7alpha,8beta-dihydroxy-9alpha,10alpha-epoxy-7,8,9,10-tetrahydrobenzo[a]pyrene and K-region 4,5-epoxide exhibited moderate mutagenicity in the absence of benzo[a]pyrene-metabolizing cells. The (±)7alpha,8beta-dihydroxy-9beta,10beta-epoxy-7,8,9,10-tetrahydrobenzo[a]pyrene showed a 2000- and 270-fold higher mutation frequency for ouabain and 8-azaguanine resistance, respectively, than did the K-region 4,5-epoxide. The trans-7,8-diol which was not mutagenic in the absence of benzo[a]pyrene-metabolizing cells was more mutagenic than benzo[a]pyrene after metabolism. Mutagenesis by trans-7,8-diol in these cells was inhibited by 7,8-benzoflavone. Metabolically formed trans-7,8-diol was isolated and incubated with rat liver microsomes in the presence of co-factors. High-pressure liquid chromatography analysis indicated that the major metabolite of trans-7,8-diol is 7alpha,8beta-dihydroxy-9beta,10beta-epoxy-7,8,9,10-tetrahydrobenzo[a]pyrene. The results indicate that the latter compound is metabolically formed and the major mutagenic intermediate of benzo[a]pyrene metabolism.

3758 EXCEPTIONAL MUTAGENICITY OF A BENZO[*a*]-PYRENE DIOL EPOXIDE IN CULTURED MAMMALIAN CELLS.

(Eng.) Newbold, R. F. (Inst. Cancer Res., Pollards Wood Res. Station, Nightingales Lane, Chalfont St. Giles, Buckinghamshire HP8 4SP, U.K.); Brookes, P. *Nature* 261(5555): 52-54; 1976.

The cytotoxic and mutagenic properties of three benzo(*a*)pyrene (BP) derivatives (BP diol epoxide syn and anti configurations, and BP-4,5-oxide) were studied in V79 cells, with resistance to the purine analog 8-azaguanine used as a mutational marker. Parallel cultures of exponentially growing cells were exposed for 24 hr to a range of concentrations of each compound. Although syn and BP-4,5-oxide proved to be relatively powerful mutagens, a double logarithmic plot of mutation frequency against survival revealed that anti was over eight times as active as BP-4,5-oxide at 37% survival; anti was also much more mutagenic than syn. When sublethal doses of the anti isomer were used, mutagenesis increased proportionally with dose. The results show that this diol epoxide derivative of BP is a powerful mutagen when applied to mammalian cells, and suggest that it is the ultimate carcinogenic metabolite of BP.

3759 EFFECT OF TOBACCO SMOKE ON CILIATED LUNG EPITHELIUM OF RANA PIPIENS.

(Eng.) Kikta, M. C. (Depts. Zoology and Physiology, Ohio State Univ., Columbus, OH 43210); Holser, J. M.; Lessler, M. A. *Ohio J Sci* 76(1): 27-30; 1976.

The effects of cigarette smoke and other related ciliary toxicants, i.e., tar and nicotine on ciliated epithelium of lung tissue from the frog, *Rana pipiens*, were studied. The animals were double pithed, the lungs excised, dissected and placed in a dish of cold frog-Ringer solution. Thin slices of lung were cut off with a razor blade, and mounted inner surface up on a clean slide. Each slice of lung tissue was placed in a large drop of frog-Ringer solution (pH 7.4) containing 112.4 mM NaCl, 0.8 mM $CaCl_2$, 2.4 mM $NaHCO_3$, 0.1 mM NaH_2PO_4, 1.9 mM KCl, and 5.5 mM glucose. It was found that after solubilization of filtered cigarette smoke, by bubbling through a frog-Ringer solution, the ciliary beat and oxidative metabolism of the lung tissue decreased an average of 35% and 37%, respectively. The smoke from filtered cigarettes decreased ciliary beat 11% less than that of unfiltered cigarettes. Smoke first passed through an Aquafilter was shown to have less ciliotoxic effect with a reduction of only 12% in rate of ciliary beat as compared to the untreated control. It also was shown that cigar smoke and pipe tar residues inhibited ciliary beat to about the same degree as 0.1% nicotine.

3760 EFFECT OF AGE AND CIGARETTE SMOKING ON CARCINOEMBRYONIC ANTIGEN LEVELS.

(Eng.) Alexander, J. C., Jr. (Natl. Cancer Inst., Natl. Insts. Health, Bethesda, MD 20014); Silverman, N. A.; Chretien, P. B. *J Am Med Assoc* 235(18): 1975-1979; 1976.

Serum carcinoembryonic antigen (CEA) levels were determined by the Hansen-Z-gel technique in 276 healthy volunteers, of whom 154 were smokers and 122 were nonsmokers. The mean CEA level was significantly higher in smokers (2.7 ng/ml) than in nonsmokers (1.9 ng/ml) ($p < 0.001$), and a significantly higher percentage of smokers had elevated CEA levels ($p < 0.05$). In both groups, CEA levels were directly related to age. Seventy-six of the 154 smokers who entered the study ceased smoking. Their CEA levels were determined at 1, 3, and 6 mo after cessation of smoking. At 3 mo, elevated CEA levels declined to within the range of nonsmokers and did not appear to be influenced by previous smoking habits. Both age and smoking history must be considered for accurate evaluation of CEA levels. A reappraisal of the diseases associated with elevated CEA levels, in which the influences of age and smoking are considered, may invalidate some of the correlations previously reported. (16 references)

3761 TOBACCO AS AN AUXILIARY ETIOLOGICAL FACTOR IN CANCER OF THE LARYNX AND HYPOPHARYNX.

(Fre.) Spalajkovic, M. (No affiliation given). *J Fr Otorhinolaryngol* 25(1): 51, 52, 55; 1976.

The role of tobacco in some cancers seems well-established. The risk of lung cancer rises in direct proportion to the quantity of tobacco smoked. The same correlation may also be valid for cancers of the larynx and hypopharynx. The conclusions of research conducted on two groups of 200 patients each are reported. The first group consisted of patients with malignant tumors of the pharynx and hypopharynx. The second, or control group, was composed of persons with benign lesions of the same sites. Among the cancer group, only 13 (6%) had never smoked at all, whereas, in the second group, the corresponding figure was 77 (38%). Over 90% of the people in the cancer group smoked regularly, against 58% among the controls. All smokers inhaled the smoke. The starting age for smoking was important--those who began smoking at the earliest age ran the greatest danger. The length of intoxication, i.e., the number of years of uninterrupted smoking, also increased the risk, as did the number of cigarettes. The mortality rate from cardiovascular or respiratory illnesses was markedly higher among smokers than among nonsmokers. Besides its potential as a carcinogen, tobacco smoke undermined the local immunity of the respiratory tract, which led to infections.

3762 REGULATION OF THE ACTIVITY OF MICROSOMAL ENZYMES BY PHOSPHOLIPIDS IN VIVO.

(Rus.) Burlakova, E. B. (Inst. Chemical Physics, Acad. Sciences USSR, Moscow, USSR); Dzhaliabova, M. I.; Molochkina, E. M. *Dokl Akad Nauk SSSR* 227(4): 991-994; 1976.

Changes in the oxidation intensity in lipids and changes in the phospholipid concentration were studied in the endoplasmic reticulum of mouse liver microsomes following treatment with 3,4-benzpyrene and the antioxidants 4-hydroxy-3,5-di-tert-butyl-α-methylbenzylamine and 6 methyl-2-ethyl-3-hydroxypyridine and also during Ehrlich ascites tumor development in mice. Glucose-6-phosphatase (G-6-Pase) was used to indicate the functional activity of the microsomal membrane. In experiments using the cancer cells or the antioxidants, the antioxidative activity of lipids and G-6-Pase activity increased. 3,4-Benzpyrene decreased both lipid antioxidative and G-6-Pase activity. A correlation between the oxidation level in lipids and the functional activity of the membranes was established. A correlation between G-6-Pase activity and phosphatidylethanolamine levels confirmed the effector role of the amine in G-6-Pase reactions in vivo and in vitro. Oxidation in lipids in vivo regulates the activity of membrane-bound enzymes through changes in the concentration of phospholipids serving as effectors for enzymes.

3763 METABOLIC ACTIVATION OF AROMATIC HYDROCARBONS IN PURIFIED RAT LIVER NUCLEI: INDUCTION OF ENZYME ACTIVITIES AND BINDING TO DNA WITH AND WITHOUT MONOOZYGENASE-CATALYZED FORMATION OF ACTIVE OXYGEN.

(Eng.) Rogan, E. G. (Eppley Inst. Res. Cancer, Univ. Nebraska Medical Center, Omaha, NB 68105); Mailander, P.; Cavalieri, E. *Proc Natl Acad Sci USA* 73(2): 457-461; 1976.

Metabolism of polycyclic aromatic hydrocarbons by microsomal monooxygenases as the cellular method for detoxification and disposal of these compounds was examined. Purified rat liver nuclei covalently bound low levels of seven aromatic [^{14}C]hydrocarbons to nuclear DNA. Induction of monooxygenases with 3-methylcholanthrene increased the binding of six carcinogenic hydrocarbons, but did not raise the level of binding of noncarcinogenic anthracene. Removal of the nuclear envelope by Triton N-101 eliminated binding and aryl hydrocarbon hydroxylase activities and cytochrome P-450 from the nuclei. Binding of two strong carcinogens, benzo[a]pyrene and 7,12-dimethylbenz[a]anthracene, to nuclear DNA was compared to the levels of aryl hydrocarbon hydroxylase and cytochrome P-450 in nuclei from uninduced and benz[a]anthracene-, 3-methylcholanthrene- and phenobarbital-induced rat monooxygenases. Microsomal hydroxylase and cytochrome P-450 were also assayed. Induction with 3-methylcholanthrene gave the largest increases in nuclear activities: 11 times as much hydroxylase, 6 times as much cytochrome P-450, and 4 times as much binding of both hydrocarbons. Benz[a]anthracene and phenobarbital induced these nuclear activities 0- to 4-fold. In the presence of added NADPH, binding of benzo[a]pyrene to DNA by nuclei increased rapidly for at least 20 min. When NADPH was not added, the reaction stopped at a low level in 5 min. When CO was bubbled through the reaction mixture with or without added NADPH, binding of benzo[a]pyrene and 7,12-dimethylbenz[a]anthracene was partially inhibited, indicating that cytochrome P-450 plays a role in this activation. That activation and subsequent binding of hydrocarbons to nuclear DNA, at least in part, does not require the activated oxygen used in monooxygenase reactions was demonstrated since no nuclear hydroxylase activity was seen without added NADPH or in the presence of CO.

3764 TISSUE DISTRIBUTION, BINDING PROPERTIES AND LESIONS PRODUCED BY DEHYDRORETRONECINE IN THE NONHUMAN PRIMATE.

(Eng.) Hsu, I. C. (Medical Sch., Univ. Wisconsin, Madison, WI 53706); Robertson, K. A.; Allen, J. R. *Chem Biol Interact* 12(1): 19-28; 1976.

Four-month-old rhesus monkeys were injected with 65 mg of [^{3}H]dehydroretronecine per kg and sacrificed at 6, 12 and 24 hr following injection. By the 24th hr, 13% of the dose had been eliminated in the urine. Although there were no feces, the extremely high radioactivity of the bile and intestinal contents suggested that this route was a major one for the excretion of this compound. The ^{3}H was distributed throughout the body by the 6th hr (with the greatest percentage being in the skin and muscle). It was in the gastric mucosa that the lesions produced by dehydroretronecine were the most severe. High levels of radioactivity persisted in the gastric mucosal lysates after washings with trichloroacetic acid, while only a small percentage of the ^{3}H remained in the washed liver lysate. Over 20% of the ^{3}H was bound to mucosal protein and less than 1% to the nucleic acids.

3765 THE ROLE OF PROLACTIN ON BREAST CANCER. PART 2. THE ROLE OF PROLACTIN ON SHIONOGI CARCINOMA-115.

(Jpn.) Iwado, M. (Okayama Univ. Medical Sch., Okayama, Japan). *Okayama Igakkai Zasshi* 88 (1, 2): 111-117; 1976.

In part 1, it was established that breast cancer does not occur in the absence of prolactin. In this study, prolactin concentrations in experimental and control groups of male mice were studied by disk electrophoresis. The experimental groups had received artificial transplants of Shionogi carcinoma-115 (a testosterone-dependent tumor) in their hypophysis. One control group received transplants of Shionogi carcinoma-42 (a testosterone-independent tumor) in their hypophysis. The other control group did not receive transplants. After transplantation, prolactin or testosterone was administered to each group on days 10 and 14. It was found that the concentration of prolactin in the Shionogi carcinoma-115 group was approximately four times higher than in the nontumor control group and approximately three times higher than in the other control group (Shionogi carcinoma-42). The P uptake study indicated that the hormonal dependence of Shionogi carcinoma-115 could be altered by administration of prolactin or testosterone and that the dependence could be recovered by the second or third tumor transplants. The administration of prolactin or testosterone increased P uptake 6 to 6.5 times over the control group (without any administration). Generally, male mice are considered to have low secretions of prolactin. Therefore, it is noteworthy that an increase in prolactin secretion is accompanied by the occurrence of a tumor, which indicates a direct relationship. (18 references)

3766 PROTECTIVE EFFECT OF GLYCYRRHIZIN IN LIVER INJURY INDUCED BY A CARCINOGEN (THE FIRST REPORT) (Meeting Abstract). (Eng.) Watari, N. (First Dept. Anatomy, Nagoya City Univ. Medical Sch., Nagoya, 467 Japan). *J Cell Biol* 70(2/Part 2): 1a; 1976.

3767 LINOLEIC ACID HYDROPEROXIDE--HEME COMPOUND ACTIVATION OF THE CARCINOGEN N-HYDROXY-2-ACETYLAMINOFLUORENE. INHIBITION BY ASCORBATE (Meeting Abstract). (Eng.) Floyd, R. A. (Biomembrane Res. Lab., Oklahoma Medical Res. Foundation, Oklahoma City, OK 73104); Soong, L.; Stuart, M.; Walker, R. N. *Biophys J* 16(21-part 2): 55a; 1976.

3768 USE OF WATER SLURRIES IN AFLATOXIN ANALYSIS. (Eng.) Velasco, J. (U.S. Dept. Agriculture, Agricultural Res. Service, Agricultural Marketing Res. Inst., Beltsville, MD 20705); Morris, S. L. *J Agric Food Chem* 24(1): 86-88; 1976.

3769 TOXICITY TO WHITE MICE OF CORN NATURALLY CONTAMINATED WITH AFLATOXIN. (Eng.) Lindenfelser, L. A. (Res. and Development Div., Commercial Solvents Corporation, Terre Haute, IN 47808); Lillehoj, E. B.; Sansing, G. A. *J Agric Food Chem* 24(1): 198-200; 1976.

3770 RADIOACTIVITY IN URINE AND FECES OF MINK (*MUSTELA VISON*) TREATED WITH [^{14}C] AFLATOXIN B_1. (Eng.) Chou, C.-C. (Dept. Food Science, Food Res. Inst., Univ. Wisconsin-Madison, Madison, WI 53706); Marth, E. H. *Arch Toxicol (Berl)* 35(2): 75-81; 1976.

3771 IN VITRO ACTIVATION OF AFLATOXIN B_2 TO METABOLITES MUTAGENIC IN *NEUROSPORA CRASSA* (Meeting Abstract). (Eng.) Matzinger, P. K. (Natl. Inst. Environmental Health Sciences, Res. Triangle Park, NC); Ong, T.-M. *Mutat Res* 38(2): 121; 1976.

3772 ENDOPLASMIC RETICULUM LAKES AND MALIGNANT HEPATOMA (Letter to Editor). (Eng.) Theron, J. J. (Inst. Cellular Physiology, Univ. Pretoria, Pretoria, South Africa); van der Watt, J. J. *S Afr Med J* 50(12): 461; 1976.

3773 INFLUENCE OF RIBOFLAVIN ON DISTURBANCES IN TRYPTOPHAN METABOLISM AND HEPATOMA PRODUCTION AFTER A SINGLE DOSE OF AFLATOXIN B_1. (Eng.) Lemonnier, F. J. (Group U 56 I.N.S.E.R.M., Hopital de Bicetre, F-94270 Kremlin-Bicetre, France); Scotto, J. M.; Thoung-Trieu, C. *J Natl Cancer Inst* 55(5): 1085-1087; 1975.

3774 PRELIMINARY REPORT ON AN UNEXPECTED EFFECT OF AN EXTRACT FROM *DRYOPTERIS FILIX MAS*. (Ger.) Kantemir, I. (Ankara Universitesi Tip Fakultesi (Sihhye), Farmakoloji Enstitusu, Ankara, Turkey); Akder, G.; Tulunay, O. *Arzneim Forsch* 26(2): 261-262; 1976.

3775 ON THE ACTIVE PRINCIPLES OF THE SPURGE FAMILY. III. SKIN IRRITANT AND COCARCINOGENIC FACTORS FROM THE CAPER SPURGE. (Eng.) Adolf, W. (Institut fuer Biochemie, Deutsches Krebsforschungszentrum, Heidelberg, West Germany); Hecker, E. *Z Krebsforsch* 84(3): 325-344; 1975.

3776 INFORMOFER PROTEINS: THE TARGET MOLECULES OF AZOCARCINOGENS IN LIVER CELLS (Meeting Abstract). (Eng.) Holoubek, V. (Dept. Human Biological Chemistry and Genetics, Univ. Texas Medical Branch, Galveston, TX 77550); Patel, M. T.; Yoshida, M. *J Cell Biol* 70(2/Part 2): 88a; 1976.

3777 STUDIES ON CARCINOGENIC AZO DYES. VI. EFFECT OF FACTORS INFLUENCING DRUG METABOLISM ON THE NIH SHIFT DURING HYDROXYLATIONS OF 4-DIMETHYLAMINO-3'-METHYLAZOBENZENE AND 3-METHYLACETANILIDE. (Eng.) Mori, Y. (Gifu Coll. Pharmacy, Mitahora higashi 5-6-1, Gifu, Japan); Toyoshi, K.; Baba, S. *Chem Pharm Bull (Tokyo)* 24(3): 500-506; 1976.

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3826 MUTAGENIC EFFECTS OF VINYL CHLORIDE IN *DROSOPHILA MELANOGASTER* (Meeting Abstract). (Eng.) Magnusson, J. (Wallenberg Lab., Univ. Stockholm, Stockholm, Sweden); Ramel, C. *Mutat Res* 38(2): 115; 1976.

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3829 ATTEMPTS TO DETECT *AGROBACTERIUM TUMEFACIENS* DNA IN CROWN GALL TUMOR TISSUE. (Eng.) Merlo, D. J. (Dept. Microbiology and Immunology, Univ. Washington, SC-42, Seattle, WA 98195); Kemp, J. D. *Plant Physiol* 58(1): 100-106; 1976.

See also:

* (Rev): 3601, 3602, 3603, 3604, 3605, 3606, 3607, 3608, 3609, 3610, 3611, 3612, 3613, 3614, 3615, 3616, 3617, 3618, 3619, 3620, 3621, 3622, 3623, 3624, 3625, 3626, 3627, 3628, 3671, 3701, 3702, 3703, 3704, 3705, 3706, 3707, 3708, 3709, 3710, 3711, 3712, 3713, 3714, 3715, 3716, 3725, 3727, 3734
* (Phys): 3856, 3857, 3869, 3878, 3881, 3883
* (Viral): 3953
* (Immun): 4028, 4054
* (Path): 4106
* (Epid-Biom): 4121, 4122, 4127, 4128, 4129, 4130, 4131, 4135, 4136, 4138, 4145

3830 MULTIPLE SIMULTANEOUS EVENT MODEL FOR RADIATION CARCINOGENESIS.

(Eng.) Baum, J. W. (Health Physics and Safety Div., Brookhaven Natl. Lab., Upton, NY 19973). *Health Phys* 30(1): 85-90; 1976.

A mathematical model was proposed that postulated that cancer induction was a multi-event process, that these events occurred naturally, usually one at a time in any cell, and that radiation frequently caused two of these events to occur simultaneously. Microdosimetric considerations dictated that for high linear energy transfer radiations the simultaneous events were associated with a single particle or track. The model predicted linear dose-effect relations for early times after irradiation with small doses, approximate power functions of dose having exponents less than 1 for populations of mixed age examined at short times after irradiation with small doses, saturation of effect at either long times for irradiation with small doses or for all times after irradiation with large doses, and a net increase in incidence dependent on age of observation but independent of age at irradiation. The model provided a quantitative framework to explain unexpected results. It also provided a logical framework to explain the dose-effect relations observed in the Japanese survivors of the atomic bombs.

3831 DOSE-RESPONSE CURVES AND THEIR MODIFICATION BY SPECIFIC MECHANISMS.

(Eng.) Yuhas, J. M. *In: Biology of Radiation Carcinogenesis*. Edited by J. M. Yuhas; R. W. Tennant; J. D. Regan. (New York: Raven Press): pp. 51-61; 1976.

At least three mechanisms can contribute to the yield of radiation induced cancers, the relative contribution of each depending heavily on exposure conditions and the tumor type involved. The existence of different mechanisms is evidenced by a discontinuity in the dose-response curve for papillary lung adenomas in mice treated with increasing doses of localized x-rays. At low doses (250-750 rads) the dose-response curve is linear, indicating that a single ionizing event is responsible for tumor induction. At higher doses (750-[illegible]00 rads) an increased slope indicates an additional contributing mechanism, namely, the interaction of ionizations within the target cells, each of which was by itself unable to produce a tumor. The induction of murine papillary lung adenomas by urethan demonstrates a dose-response curve similar to that with x-rays. This need not result from similar processes; the event interaction in the curvilinear region could reflect immunosuppression or prolonged retention time that does not occur with low doses. The administration of 600-700 rads as four weekly fractions is more leukemogenic in C57BL mice than is the acute administration of the same dose. This may be consistent with the observation that chronic radiation induces endogenous leukemic viruses more effectively than acute irradiation. It is proposed that radiation increases leukemic incidence either by inducing such a heavy viral burden that immune responsiveness is irrelevant, or by suppressing immune responsiveness so that even small viral burdens can prove to be significant. Experiments designed to test these proposals are described. (18 references).

3832 CHROMOSOMAL ABERRATIONS IN PERSONS OCCUPATIONALLY EXPOSED TO IONIZING RADIATION.

(Cro.) Horvat, D. (Institut za medisinska istrazivanja i medicinu rada JAZU, Zagreb, Yugoslovia). *Arh Hig Rada Toksikol* 26(2): 139-146; 1975.

The objective of this study was to find out the mutagenic effects of ionizing radiation on persons occupationally exposed to it. X-ray technicians, radiologists, and physiologists, two of each, as well as four secretaries working in radiology, comprised the test group. For 6 mo prior to the experiment no subject took drugs that could eventually cause chromosomal changes, nor were they irradiated for diagnostic purposes or therapy. The control group of 10 persons had never been exposed to ionizing radiation professionally, nor did they use any mutagenic agents. Furthermore, they did not suffer from any illness in the 6 mo prior to this study. Chromosomal analyses of lymphocyte cultures were performed by the Moorhead technique. Metaphases of each member of the test (50-100 metaphases) and control groups (50) were analyzed. The results show that the predominant lesions in the exposed group were chromosome breaks, acentric and bicentric chromosomes, and polyploidy in 3% of cases. In over 1% of cases the translocations appeared to be permanent chromosomal changes. Compared with the controls, the rate and type of chromosomal changes in exposed subjects were significantly higher.

3833 ONCOGENIC TRANSFORMATION IN VITRO BY X-RAYS: INFLUENCE OF REPAIR PROCESSES.

(Eng.) Terzaghi, M.; Little, J. B. *In: Biology of Radiation Carcinogenesis*. Edited by J. B. Yuhas, R. W. Tennant, and J. D. Regan. (New York: Raven Press): pp. 327-334; 1976.

X-ray induced oncogenic transformations in a C3H mouse embryo derived cell line appeared to be produced by a specific effect of the radiation on the cells rather than by an effect mediated by nonspecific cell killing. There was exponential increase in transformants per surviving cell doses between 50 and 400 rads. At doses above 400 rads delivered to exponentially growing cells, the transformation frequency remained at approximately two transformants/10^3 surviving cells. Little decline in survival occurred at doses below 300 rads. Survival declined exponentially at higher doses with a D_0 of 180 rads. When cells were irradiated in density-inhibited plateau-phase growth and allowed to repair potentially lethal radiation damage (subculture was delayed for 3 hr postirradiation), the D_0 of the survival curve increased to 250 rads. Under these conditions, the transformation frequency continued to increase exponentially with doses up to 1,000 rads. These results indicate that the processes controlling cell survival and transformation are related and that molecular repair mechanisms may be intimately involved in both effects.

3834 OBSERVATIONS ON POLYCYTHEMIA VERA TURNING INTO ACUTE OR CHRONIC GRANULOCYTIC LEUKEMIA DURING TREATMENT WITH RADIOACTIVE PHOSPHORUS ^{32}P. (Eng.) Krasnik, W. (Province Outpatient Clinics, Poznan, Poland). *Pol Med Sci Hist Bull* 15/3(4): 447-451; 1975.

Three cases of acute granulocytic leukemia that developed in a series of 172 patients with polycythemia vera treated with radioactive phosphorus (^{32}P) are briefly described, and another six cases of chronic granulocytic leukemia are cited. The group comprised 74 men and 98 women seen over a 14-yr period. ^{32}P was generally given po and only exceptionally iv. One patient (50 yr old) received 3 mCi of ^{32}P; a 2-yr remission followed. During the next 11 yr, six po doses were given (total, 47.7 mCi); 18 mo after the last dose signs of hemorrhagic diathesis developed, and cells resembling myeloblasts were found in the peripheral blood. Survival time from diagnosis was 16 yr and from onset of ^{32}P treatment was 11.5 yr. In a 48-yr-old patient, ^{32}P (4.5 mCi) was first given iv 4 yr after diagnosis of polycythemia; three more doses were given for a total of 19 mCi. Acute granulocytic leukemia developed after the last dose at the peak of a 2-yr remission. Only mononuclear atypical cells were seen in the peripheral blood and bone marrow smears. In the third case (50 yr old), two 3.5-mCi doses were given iv 6 mo apart. Acute granulocytic leukemia appeared 4 wk after the second dose, and the patient died. In peripheral blood smears and in the bone marrow only atypical immature cells were seen. The six cases of chronic granulocytic leukemia developed slowly over 2-3 yr with episodes of regression. It is concluded that the development of acute granulocytic leukemia during treatment with ^{32}P is the result of the leukogenic action of ionizing radiation. Also, the transformation of polycythemia vera into chronic granulocytic leukemia seems to be the natural outcome of this disease in patients in whom ^{32}F treatment resulted in prolonged survival.

3835 REPAIR OF DNA STRAND BREAKS IN CHINESE HAMSTER CELLS (Meeting Abstract). (Eng.) Dugle, D. L. (Medical Biophysics Branch, Whiteshell Nuclear Res. Establishment, Atomic Energy Canada Ltd., Pinawa, Manitoba, Canada); Gillespie, C. J.; Chapman, J. D. *Radiat Res* 67(3): 513; 1976.

3836 REPAIR OF STRAND BREAKS IN MAMMALIAN DNA: THROUGHOUT THE CELL CYCLE (Meeting Abstract). (Eng.) Sun, C. (Dept. Radiology and Radiation Biology, Colorado State Univ., Fort Collins, CO 80523); Lett, J. T. *Radiat Res* 67(3): 586; 1976.

3837 ATP REQUIREMENTS FOR THE REPAIR OF X-RAY INDUCED DNA DAMAGE IN CULTURED MOUSE L-299 CELLS (Meeting Abstract). (Eng.) Nagle, W. A. (Div. Nuclear Medicine and Radiation Biology, Univ. Arkansas for Medical Sciences, Little Rock, AR 72206); Moss, A. J.; Baker, M. L. *Radiat Res* 67(3): 575; 1976.

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See also:

* (Rev): 3619, 3624, 3649, 3659, 3660, 3661, 3662, 3663, 3664, 3665, 3701, 3704, 3706, 3724
* (Chem): 3755, 3792
* (Viral): 3912, 3913
* (Immun): 4044
* (Epid-Biom): 4121, 4131, 4137, 4139

3891 SYNTHESIS OF AVIAN RNA TUMOR VIRUS STRUCTURAL PROTEINS. (Eng.) Eisenman, R.; Vogt, V. M.; Diggelmann, H. *In: Tumor Viruses. Cold Spring Harbor Symposia on Quantitative Biology.* Cold Spring Harbor Lab. (Cold Spring Harbor, NY): vol. 39, pp. 1067-1075; 1975.

The synthesis of avian RNA tumor virus structural proteins from the precursor was investigated. Study of the kinetics of the labeling of intracellular avian myeloblastosis virus (AMV) internal proteins VP24 and VP11 in AMV-infected cells during pulse-chase experiments showed that the unstable virion polypeptides Pr76, Pr66, Pr60, Pr32, and Pr12 are precursors to virion proteins. Cation exchange chromatography experiments showed that the methionine-containing sequences of virion proteins VP19, VP24, and VP11 were contained in Pr76. Pr66 was generated by cleavage of VP11 from Pr76, and Pr60 was probably derived from Pr66 by splitting off of residues not containing methionine. Pr32 appeared to lack the peptides corresponding to VP24 and the unassigned peak k, while all four peaks of VP19 were present. A new peptide eluting behind the major VP24 peptide could represent an altered form of peak k. Avian oncornaviruses also contain two other internal proteins, VP12 and VP10, the former also contained in Pr76. The drug pactamycin was used to determine the order of proteins on Pr76 in AMV-infected chick cells. Pactamycin preferentially reduced the incorporation of methionine label into VP19 and VP24 with respect to VP11. Thus, the order of methionine-labeled proteins from the N-terminal end of the precursor appears to be VP19-VP24-VP11. Furthermore, the cleavage of structural proteins appears to commence only on completed, as opposed to nascent, precursor molecules. The cells of the nonproducing avian oncornavirus-transformed cell line, H-RSV(BH), although capable of synthesizing the polypeptide that is a precursor to viral structural proteins, did not generate these proteins by subsequent proteolytic cleavage. However, under conditions leading to rescue of virus particles, precursor molecules were synthesized and cleaved to form the virion proteins as in productively infected cells. None of a variety of other mammalian cells transformed by the avian RNA tumor viruses showed the presence of polypeptides having the electrophoretic mobility of any known virion or virion precursor proteins. (31 references)

3892 EFFECT OF IN VITRO ADAPTATION OF MAREK'S DISEASE VIRUS ON POCK INDUCTION ON THE CHORIOALLANTOIC MEMBRANE OF EMBRYONATED CHICKEN EGGS. (Eng.) Sharma, J. M. (Agriculture Res. Service, Regional Poultry Res. Lab., East Lansing, MI 48823); Coulson, B. D.; Young, E. *Infect Immun* 13(1): 292-295; 1976.

A study was conducted to determine if tissue culture adaptation of Marek's disease virus would affect its ability to induce pocks on the chorioallantoic membrane of embryonated chicken eggs and to induce plaque formation in duck embryo fibroblasts. Duplicate cultures of 24-hr-old secondary duck embryo fibroblast cell cultures were inoculated with each 10-fold serial dilution of cell-associated virus isolate, using 0.1 ml inoculum per culture. Plaques were enumerated 12-14 days after inoculation. A portion (0.1 ml) of each virus dilution was inoculated iv into each of 10 11-day-old chicken embryos. After 5 days at 37 C, the embryos were chilled at 4 C overnight and observed for pocks. The virus preparations produced more pocks on the chorioallantoic membrane of embryonated chicken eggs than plaques in duck embryo fibroblasts, thus indicating that lesion response in eggs was more sensitive than cytopathic response in duck embryo fibroblasts for assaying low-passage Marek's disease virus. Adaptation of the virus to cell cultures by serial passage, however, substantially reduced its pock response so that the titer ratio (plaque-forming units in duck embryo fibroblasts, pock-forming units in eggs) of cell culture-adapted Marek's disease virus was 1 or higher. The decreased pock response could not be attributed to selection of preexisting virus variant(s) with low affinity for chorioallantoic membrane because cloned Marek's disease virus had a good pock response at low cell culture passage levels, but this response decreased as the virus was attenuated by serial cell culture passage.

3893 CONTROL OF EXPRESSION OF TUMOR VIRUS GENES IN UNINFECTED CHICKEN CELLS. (Eng.) Hanafusa, H.; Hayward, W. S.; Chen, J. H.; Hanafusa, T. *In: Tumor Viruses. Cold Spring Harbor Symposia on Quantitative Biology.* Cold Spring Harbor Lab. (Cold Spring Harbor, NY): vol. 39, pp. 1139-1144; 1975.

The extent to which varying levels of viral expressions in uninfected chicken cells are determined by regulatory genes or by differences in structural genes was studied. Nucleic acid hybridization and radioimmunoassay techniques were used to determine the specific gene sequences and the synthesis of individual viral proteins. The RAV-60 virus, which has the same characteristics specified by envelope protein as the endogenous chick helper factor (chf), has been obtained from chf+ chick cells following infection with avian leukosis virus, RAV-1 or RAV-2, Bryan strain sarcoma virus, or the Schmidt-Ruppin strain of RSV. RAV-0 was isolated from line 7 chicken cells as a spontaneously produced virus. Although both RAV-0 and RAV-60 are subgroup E viruses, they have different biological and physical properties; in comparison with RAV-60, RAV-0 shows extremely poor growth in pheasant and quail cells. Sequence homologies were examined among the nucleic acids of RAV-0, RAV-2, and RAV-60 derived from RAV-2-infected chicken cells. The results showed that RAV-60 is different from RAV-0; they were consistent with the idea that RAV-60 is a recombinant between chf and exogenous leukosis virus such as RAV-2. RAV-0 DNA hybridized to a greater extent with host cell DNA than did RAV-60 or RAV-2 DNA's, supporting the interpretation that some portion of the genetic information in RAV-60 is provided by exogenous virus. The amounts of viral RNA hybridizable with the probe DNA were greatly different for chf+ and chf- cells. Thus, the viral structural genes were present in both types of cells, and it appeared that a regulatory gene controlled the transcription of these genes into viral RNA. The three major viral structural proteins, P27, P19, and P15, were detectable in all types of chick embryos, including those classified as gs-. These results suggest that the genes responsible for the synthesis of internal proteins and for the helper activity are not controlled coordinately. (30 references)

3894 TRANSFORMATION OF CELLS BY ROUS SARCOMA VIRUS: CYTOPLASMIC VACUOLIZATION. (Eng.)

Bader, A. V. (Natl. Cancer Inst., Bethesda, MD 20014); Bader, J. P. *J Cell Physiol* 87(1): 33-46; 1976.

Chick embryo cells transformed by the Bryan ''high titer'' strain of Rous sarcoma virus (RSV-BH) were heavily vacuolated. A variety of microscopic techniques were used in demonstrating that the vacuoles were cytoplasmic, bounded by membrane, and were composed largely of water. Proteins, lipids, glycoproteins, glycolipids, glycosaminoglycans, glycogen, and nucleic acids were undetectable in the vacuoles. Physiological requirements for development of the vacuoles and reversal of vacuolization, were examined in cells infected with a virus mutant, RSV-BH-Ta, which induced reversible temperature-dependent transformation. Na^+ was the only component of the cell culture medium found essential for both the development and reversal of vacuoles. Glucose depletion or dinitrophenol treatment inhibited vacuolization (suggesting a possible energy requirement in the vacuolization process. Ouabain enhanced vacuolization. Glucosamine and mannosamine prevented the disappearance of vacuoles. Cellular vacuolization could be a normal physiological response to an increase in water and Na^+.

3895 ACTIVATION AND REPRESSION OF VIRUS EXPRESSION IN MAMMALIAN CELLS INFECTED BY ROUS SARCOMA VIRUS. (Eng.)

Boettiger, D. *In: Tumor Viruses. Cold Spring Harbor Symposia on Quantitative Biology.* Cold Spring Harbor Lab. (Cold Spring Harbor, NY): vol. 39, pp. 1169-1172; 1975.

Transformed and nontransformed cells infected by Rous sarcoma virus were examined for frequency of virus rescue. It was found that nontransformed cells and revertants produced very few foci but that transformed cells produced a higher number. If infected cells contained 1 genome equivalent per cell, about 1 focus per 50 heterokaryon viral genomes was obtained. Revertants contained less than one-third genome equivalent per cell; they appear to have reverted because of a repression of viral RNA synthesis by the cell or by an alteration in viral RNA processing. Thus the control of viral expression appears to be at the level of RNA synthesis. The mechanism of virus rescue following cell fusion appears to be a provision of a cellular environment that permits progeny virus assembly.

3896 LYSIS OF ROUS SARCOMA VIRUS IN THE PRESENCE OF CHICKEN ANTIFIBROBLAST ANTISERUM AND COMPLEMENT. (Fre.)

Aupoix, M. (Unite de Virologie, I.N.S.E.R.M., 1, place du Professeur-Joseph-Renaut, 69008 Lyon, France); Huppert, J.; Vigier, P. *C R Acad Sci* [*D*] *(Paris)* 282(14): 1379-1382; 1976.

The incubation of Rous sarcoma virus (RSV) with chicken antifibroblast (CAF) antiserum, in the presence of complement, results in viral lysis. Prior research suggested the inactivation of avian viruses by CAF antiserum in the presence, but not in the absence, of complement. An experiment with RSV sought to confirm this result, using two tests: the liberation of internal viral proteins (gs) and the accessibility of viral RNA to RNase. Quantities (200 μl) of purified virus were incubated 1 hr at 37 C with 20 μl of decomplemented rabbit serum against CAF, with or without 40 μl of reconstituted hamster serum (complement). Electrophoresis disclosed five peaks, presumably corresponding to the proteins of complex gs, in the sample containing the complement but not in the sample without the complement. Therefore, incubation with chicken antifibroblast antiserum and complement liberated the internal viral proteins. Radioactive RSV was divided into three aliquots, each incubated 1 hr at 37 C. The first contained 30% Tris NaCl, the second 20% CAF antiserum and 10% Tris, the third 20% CAF antiserum and 10% complement. The two aliquots with antiserum were incubated 15 min at 37 C with 0.2 μg/ml of RNase. After the addition of RNA (270 μg/sample), SDS (1%), and pronase (1 mg/ml), the aliquots were incubated 18 hr at 4 C, then 2 hr at 37 C. The RNA extracted from each sample was precipitated and centrifuged in a glycerol gradient. In the control sample, over 50% of the labeled viral RNA appeared in the lower third of the gradient. Only 19% of the RNA could be recovered from the sample with the CAF antiserum and RNase. This could be accounted for by a partial degradation by RNase during extraction. In the sample with the antiserum, RNase, and complement, the sedimentation of RNA amounted to only 4% of the total. There remained practically no undegraded viral RNA, and the RNA under 70S had also been digested. Treatment with the CAF antiserum plus complement made most of the viral RNA accessible to RNase. The inactivation of RSV seems to demonstrate the presence of one or more antigens of the host cell in the outer viral sheath.

3897 ALL INTACT SUBUNIT RNAs FROM ROUS SARCOMA VIRUS CONTAIN POLY(A). (Eng.)

King, A. M. Q. (Univ. Wisconsin, Coll. Agricultural and Life Sciences, Madison, WI 53706); Wells, R. D. *J Biol Chem* 251(1): 150-152; 1976.

The capacity of RNA subunits of Schmidt-Ruppin Rous sarcoma virus to bind deoxythymidylate [(dT)n] was studied by cesium sulfate density gradient centrifugation, sucrose gradient centrifugation, or polyacrylamide gel electrophoresis. Two classes of subunits were observed. In agreement with previous reports, 60-70% of the purified subunits were capable of binding (dT)n. The remaining nonbinding RNAs, although indistinguishable in size from whole subunits, were shown to be fragments that had lost the ribose adenylate terminus by random nucleolytic cleavage. This was deduced from the molecular weight distribution of the nonbinding RNAs from high molecular wt RNA. In addition, virtually all subunit RNA from early harvest virus, which contains a greater proportion of intact subunit, bound (dT)n.

3898 HIGH MOLECULAR WEIGHT RNAs FROM ROUS SARCOMA VIRUS AND MOLONEY MURINE LEUKEMIA VIRUS CONTAIN TWO SUBUNITS. (Eng.) King, A. M. Q. (Univ. Wisconsin, Coll. Agricultural and Life Sciences, Madison, WI 53706). *J Biol Chem* 251(1): 141-149; 1976.

The molecular wt of the large genomic RNAs from Rous sarcoma and Moloney murine leukemia viruses were determined by a combination of sedimentation coefficients and retardation coefficients from gel electrophoresis. Six RNA standards, ranging from 0.7 x 10^6 to 5.3 x 10^6 daltons, were employed. Studies in the presence of varying concentrations of Mg^{2+} showed that the method provided valid molecular wt for RNAs of differing amounts of ordered structure. The molecular wt (x 10^{-6}) of the high molecular wt RNA complex from Rous sarcoma virus was 7.6 ± 0.3, and from murine leukemia virus was 6.9 ± 0.3. The molecular wt (x 10^{-6}) of their subunits were 3.3 ± 0.1 and 2.8 ± 0.2, respectively. Hence, the large complexes consisted to 2 subunits plus small associated RNAs. The high molecular wt RNA from cloned Rous sarcoma virus was heterogenous in molecular wt, although the apparent molecular radius was constant. Studies were performed in subfractions of the RNA as well as on RNA from virus harvested at various time intervals. The preparations with lowest molecular wt approached a mass equal to twice that of the subunit with hydrodynamic properties approaching those expected of normal single-stranded RNA. The results demonstrate that Schmidt-Ruppin RSV and Moloney MuLV both contain two RNA subunits.

3899 LOCATION OF ENVELOPE-SPECIFIC AND SARCOMA-SPECIFIC OLIGONUCLEOTIDES ON RNA OF SCHMIDT-RUPPIN ROUS SARCOMA VIRUS. (Eng.) Wang, L.-H. (Dept. Molecular Biology and Virus Lab., Univ. California, Berkeley, CA 94702); Duesberg, P. H.; Kawai, S.; Hanafusa, H. *Proc Natl Acad Sci USA* 73(2): 447-451; 1976.

A chemical method to map viral genes was applied to various avian sarcoma viruses. Envelope-specific and sarcoma-specific nucleotide sequences were located within the 10,000 nucleotides of the RNA of nondefective Schmidt-Ruppin Rous sarcoma virus (nd SR). About 30 RNase-T_1 resistant oligonucleotides were ordered relative to the 3'-poly(A)terminus of the RNA, to construct an oligonucleotide map of the nd SR RNA. A cluster of seven envelope-specific oligonucleotides, identified by their absence from an otherwise very similar oligonucleotide map of an envelope-defective deletion mutant (lacking the major viral glycoprotein), mapped at a distance of 2800-5000 nucleotides from the poly(A) end of nd SR RNA. A cluster of two sarcoma-specific oligonucleotides, identified by their absence from an otherwise nearly identical oligonucleotide map of a transformation-defective deletion mutant, mapped at a distance of 1000-2000 nucleotides from the poly(A) end of nd SR RNA. The oligonucleotide maps of nd SR and of the two deletion mutants were the same from the poly(A) end up to 650 nucleotides and included one terminal ologonucleotide, termed C (found in all avian tumor viruses tested). A postulated gene order consistent with this data suggests that sarcoma-specific nucleotide sequences map between envelope-specific nucleotide sequences and the poly(A) end of the RNA.

3900 CHARACTERIZATION AND COMPARISON OF THE MAJOR GLYCOPROTEIN FROM THREE STRAINS OF ROUS SARCOMA VIRUS. (Eng.) Krantz, M. J. (Albert Einstein Coll. Medicine, Bronx, NY 10461); Lee, Y. C.; Hung, P. P. *Arch Biochem Biophys* 174(1): 66-73; 1976.

The major glycoprotein g2 was purified from three strains of Rous sarcoma virus, representing subgroups A, B, and C. Carbohydrate analysis showed that glucosamine, mannose, galactose. fucose, and sialic acid constitute 40% of the wt of the subgroup A glycoprotein and 15% of the subgroup B and C glycoproteins. The molar ratios of the sugars were very similar. Although the amino acid compositions were also similar, they were not identical. Glycosidase digestions of subgroup A and C glycoproteins suggested the presence of two types of oligosaccharide chains, the complex serum type, with terminal sequences sialic acidα-Galβ-GlcNAcβ-, and the high mannose type with terminal α-linked mannosyl residues. After 70% of the carbohydrate was removed by glycosidases, subgroup A glycoprotein contained only glucosamine and mannose, in the molar ratio 2.0:1.3. The sequence of sugar release was consistent with oligosaccharide structures. The plant lectins concanavalin A and wheat germ agglutinin interacted strongly with the g2 glycoprotein from viruses of all three subgroups; this may be useful in purification.

3901 REVERSE TRANSCRIPTASE AND RNASE H: PRESENT IN A MURINE VIRUS AND IN BOTH SUBUNITS OF AN AVIAN VIRUS. (Eng.) Moelling, K. *In: Tumor Viruses. Cold Spring Harbor Symposia on Quantitative Biology.* Cold Spring Harbor Lab. (Cold Spring Harbor, NY): vol. 39, pp. 969-973; 1975.

The reverse transcriptase of Friend murine leukemia virus was purified and its properties were compared with the avian form of the enzyme. The sedimentation coefficient of the transcriptase was smaller than the avian form and was estimated to be 4.5S. The purified enzyme consisted of a single 8,000-dalton polypeptide that was not identical to either of the subunits of the avian enzyme. However, it is similar to the smaller subunit (α), which contains reverse transcriptase and RNase H. Regarding the avian enzyme, the α subunit was found to be a decay product of the β subunit. An attempt was made to characterize the β subunit, but it could not be purified completely; the uncleaved avian enzyme β exhibited more RNase H activity and was more efficient in transcribing natural viral RNA than the αβ complex or α alone.

3902 STUDIES ON THE ROLE OF THE HOST IMMUNE RESPONSE IN RECOVERY FROM FRIEND VIRUS LEUKEMIA: I. ANTIVIRAL AND ANTILEUKEMIA CELL ANTIBODIES. (Eng.) Chesebro, B. (Natl. Inst. Allergy and

Infectious Diseases, Rocky Mountain Lab., Hamilton, MT 59840); Wehrly, K. *J Exp Med* 143(1): 73-84; 1976.

The humoral immune response to Friend virus (FV) leukemia was studied in congenic F_1 mice differing in their incidence of recovery from leukemia. Anti-FV serum, containing FV neutralizing antibodies as well as cytotoxic antibodies for FV-induced cell surface antigens, was given to (B10.A x A.BY)F_1 mice at 1, 3, or 6 days after FV inoculation, and mice were examined for spleen foci on the 9th day. Anti-FV serum given 1 or 3 days after virus reduced the number of virus-induced spleen foci. Antiserum given on day 6 was not effective. Anti-viral neutralizing antibodies rose in titer in vivo concurrently with disappearance of viremia and fall in spleen virus levels. Cytotoxic antileukemia cells antibodies also appeared at this time. Passive transfer of these antibodies could inactivate low numbers of leukemia cells in vivo; however, mice of both high and low recovery genotypes produced antibodies in equal titer and recovered from viral infection in spite of striking differences in recovery from leukemic splenomegaly. Female (B10.A x A.BY)F_1 and (C57BL/10 x A.BY)-F_1 mice were inoculated iv with 15,000 focus forming units of B-tropic FV, and were subsequently examined for viremia and antibodies. Mice lacking C57BL genes did not produce antibodies or recover from viremia except in rare instances. Recovery from splenomegaly was found to be influenced by 3 or more C57BL genes independent of the H-2 complex. The data suggest that neutralizing anti-FV antibody may play a major role in elimination of viral infection, and that this may be a necessary, but not alone sufficient, step in the process of recovery from FV-induced, splenomegaly.

3903 CHANGES IN RNA AND PROTEIN METABOLISM PRECEDING ONSET OF HEMOGLOBIN SYNTHESIS IN CULTURED FRIEND LEUKEMIA CELLS. (Eng.) Sherton, C. C. (Univ. Oregon Health Sciences Center, Portland, OR 97201); Kabat, D. *Dev Biol* 48(1): 118-131; 1976.

The hematopoietic process induced by dimethyl sulfoxide (Me_2SO) in the Ostertag FSD-1 line of Friend erythroleukemia (FL) cells and the changes preceding the onset of Hb synthesis at 48 hr were studied. Although cellular viability, mitotic rate, and DNA content were unaffected by the induction procedure, the induced cells became progressively smaller, and by 96 hr contained only 55% as much RNA and 60-70% as much protein as control cells. The decline in RNA content was signficantly by 24 hr and affected 4S and ribosomal RNA in a noncoordinated manner throughout the hematopoietic process. Incorporation of radioactive uridine into the 45S precursor of ribosomal RNA was specifically inhibited by 1-2 hr after first treating FL cultures with 1% Me_2SO. This earliest known effect of Me_2SO on FL cells was followed by a decline in synthesis of protein. The basic sequence of macromolecular and cell size changes were similar to those occurring during normal erythropoiesis.

3904 MEMBRANE ACTION OF DMSO AND OTHER CHEMICAL INDUCERS OF FRIEND LEUKAEMIC CELL DIFFERENTIATION. (Eng.) Lyman, G. H. (Dept. Medicine, Roswell Park Memorial Inst., 666 Elm St., Buffalo, NY 14263); Preisler, H. D.; Papahadjopoulos, D. *Nature* 262(5567): 360-363; 1976.

The growth of Friend leukemic cells (FLC) in suspension culture in the presence of dimethyl sulfoxide (DMSO) and other cryoprotective agents results in the differentiation of these cells along the erythroid pathway. These agents increased the normal phase transition temperature of the FLC phospholipids and produced a new transition at high temperatures, suggesting increased membrane stability and decreased membrane fluidity. Local anesthetics, agents known to increase membrane fluidity, inhibit the differentiation of FLC cells. When the cells were grown in the presence of both anesthetics and DMSO, this inhibition was abolished. Divalent cations such as Ca^{+2} and Mg^{+2} increase the stability of membranes and induce FLC differentiation. The combination of divalent cations with DMSO increased FLC differentiation and decreased anesthetic-induced inhibition. It is suggested that DMSO acts at the level of the cell membrane to induce differentiation by increasing membrane stability. This increased stability could result in membrane changes that initiate transcription of the messenger RNA associated with differentiation. (22 references)

3905 ANTIGENIC RELATIONSHIPS BETWEEN MURINE, FELINE AND PRIMATE RNA TUMOR VIRUSES AND MEMBRANE ANTIGENS OF HUMAN LEUKEMIC CELLS. (Eng.) Metzgar, R. S.; Mohanakumar, T.; Bolognesi, D. P. *In: Comparative Leukemia Research.* Edited by J. Clemmesen and D. S. Yohn. (Basel, Karger): 549-554; 1976.

Data are presented on the cytotoxicity reactions of antiserum to RNA tumor virus antigens with human leukemic cells. The Friend leukemic virus (FLV) gp71 antiserum reacted only with cells from patients with chronic myelogenous leukemia (CML) or acute myelomonocytic leukemia (AMML). However, FLV antiserum additionally reacted with cells from all acute myelogenous leukemia and AMML patients plus 1/26 chronic lymphocytic leukemia (CLL) and 3/10 acute lymphocytic leukemia (ALL) patients. Goat antiserum to feline leukemic virus failed to react with normal peripheral blood lymphocytes, but did react with cells from many patients with all morphological types of leukemia. The goat antiserum to simian sarcoma virus (SSV-1) failed to react with cells from CML patients but reacted with those from 18/21 patients with CLL and 8/10 ALL samples. Similar cytotoxic activity was noted with the bovine and goat antiserum to Gibbon ape lymphoma virus. Some preliminary results of absorption studies of the various antisera with cells from various leukemic patients were included.

3906 FURTHER CHARACTERIZATION OF THE FRIEND MURINE LEUKEMIA VIRUS REVERSE TRANSCRIPTASE-RNase H COMPLEX. (Eng.) Moelling, K. (Max-Planck-Institut fuer Molekulare Genetic, D-1 Berlin-33, West Germany). *J Virol* 18(2): 418-425; 1976.

To determine whether the Friend murine leukemia enzyme (84,000 molecular wt) exhibits the properties of a true viral reverse transcriptase-RNase H complex, several of its enzymatic properties were compared to those of the avian α subunit, as representative of a degraded enzyme, and to the $\alpha\beta$ avian enzyme complex. The intact murine enzyme resembled the avian β-containing enzyme complex and was different from α in the following respects: (a) it bound to native RNA templates; (b) it transcribed native RNA templates into DNA, a reaction that can be inhibited by actinomycin D; (c) RNase H activity behaved like a processive exonuclease; and (d) analysis of the RNase H digestion products revealed oligonucleotides approximately four bases in length. These results indicate that this polypeptide retains all the properties known for the reverse transcriptase-RNase H complex of the avian viruses. The properties are: the ability to bind to natural RNA and to transcribe it without the addition of synthetic fibers; inhibition of DNA synthesis by actinomycin D; copurification of an RNase H; exonucleolytic mode of action of the RNase H activity; and oligomers, approximately tetranucleotides, as the main RNase H digestion products and the absence of mononucleotides.

3907 RELATIONSHIP OF INFECTIOUS MURINE LEUKEMIA VIRUS AND VIRUS-RELATED ANTIGENS IN GENETIC CROSSES BETWEEN AKR AND THE FV-1 COMPATIBLE STRAIN C57L.

(Eng.) Ikeda, H. (Asahikawa Medical Coll., Asahikawa, Japan); Rowe, W. P.; Boyse, E. A.; Stockert, E.; Sato, H.; Jacobs, S. *J Exp Med* 143(1): 32-46; 1976.

In a genetic study of murine leukemia virus (MuLV) and its components, the backcross C57L x (C57L x AKR) was examined. This population was selected because strains AKR and C57L were both Fv-1n, and the restriction that the Fv-1b allele imposed on the output of virus was obviated. The segregants were scored for three characteristics: infectious Gross-AKR-MuLV (V), in the tail; group-specific antigen indicative of p30 internal viral protein, in spleen; and GIX antigen, now thought to be indicative of gp69/71 viral envelope glycoprotein, on thymocytes. It was confirmed that the AKR mouse had two unlinked chromosomal genes, Akv-1 and Akv-2, each of which could independently give rise to the life-long high output of MuLV characteristic of AKR mice. Of the 8 phenotypes that could possibly be derived from segregation of the 3 pairs of independent alternative traits, 7 were observed, but on progeny testing only 3 were shown to reflect stably heritable genotypes; these were V+p30+GIX+ and V-p30-GIX- (the parental types) and V-p30+GIX+. A third, newly identified AKR gene, designated Akvp, segregated independently of Akv-1 and Akv-2, also determined expression of p30 and GIX but in this case independently of XC-detectable MuLV.

3908 EFFECT OF RAUSCHER LEUKEMIA DEVELOPMENT ON DNA SYNTHESIS BY HEMATOPOIETIC CFU-S.

(Eng.) Okunewick, J. P. (Allegheny General Hosp., 320 East North Ave., Pittsburgh, PA 15212); Phillips, E. L.; Brozovich, B. *Exp Hematol* 4(3): 143-150; 1976.

Experiments were conducted to determine if ip injections of Rauscher leukemia virus altered the proportion of pluripotent hematopoietic colony forming units in the spleen (CFU-S) in DNA synthesis, initially and then as the disease developed. The effect of the viral leukemia on the number of CFU-S was measured as a function of time after virus administration. The CFU-S in DNA synthesis increased substantially after the first week, reaching a maximum at 2 wk, after which they decreased, as determined by the in vitro ^{3}HTdR suicide technique. Within 4 hr of virus injection, the number of spleen CFU-S inactivated by in vitro ^{3}HTdR exposure increased to 20.9% from a normal mean value of 10.2%. For the next 2 wk the proportion of CFU-S responding to tritiated thymidine exposure was approximately 20%; the change in number of surviving CFU-S roughly paralleled the change in total colony forming cells. In the third week after injection the proportion of CFU-S inactivated by ^{3}HTdR decreased and could no longer be detected, even though the total number of CFU-S was elevated severalfold over the normal. These findings indicate that infection with moderate dose levels of Rauscher virus results in a measurable early increase in the proportion of cells in DNA synthesis. However, further substantial increases could not be detected as the disease developed, suggesting that the virus itself does not initiate all the steps of DNA synthesis. Rather, it appears to be dependent to some degree upon other aspects of normal DNA synthesis that remain under control of the cell.

3909 MODIFICATION OF HEMOPOIETIC STEM CELLS OF BALB/C MICE BY RAUSCHER LEUKEMIA VIRUS.

(Eng.) Van Beek, H. J. (Dept. Experimental Pathology, Erasmus Univ., Rotterdam, The Netherlands); Van't Hull, E.; Van Griensven, L. J. L. D. *Exp Hematol* 4(3): 151-160; 1976.

To investigate the nature of hemopoietic stem cells, bone marrow and spleen from Rauscher leukemia virus (RLV)- infected BALB/c mice were tested for proliferative activity at various times after infection by spleen colony assay. Colonies derived from both bone marrow and spleen showed a modified maturation behavior. The erythroid colonies were classified into three consecutive stages of maturation according to morphologic criteria. Colonies derived from both types of cells exhibited maturation retardation upon RLV infection of the donor mice; the spleen-derived erythroid colonies showed the most severe changes. RLV itself could not mimic this phenomenon; moreover, as shown by immunofluorescence, only low amounts of RLV were present in the spleen colonies. It is therefore suggested that RLV modifies part of the stem cell compartment, leading to a prolonged cycle time of proerythroblasts and hence to a retardation of colony growth.

3910 STUDIES ON THE DISTRIBUTION AND ORIGIN OF CYTOLYTIC T LYMPHOCYTES PRESENT IN MICE BEARING MOLONEY MURINE SARCOMA VIRUS (MSV)-INDUCED TUMORS.

(Eng.) Plata, F. (Ludwig Inst. Cancer Res., Bugnon 21, CH-1011, Lausanne, Switzerland); MacDonald, H. R.; Sordat, B. *Bibl Haematol (Basel)* (43): 274-277; 1976.

C57BL mice inoculated with Moloney sarcoma virus (MSV) developed palpable tumors on day 4, with maximum tumor size occurring between days 9-11, followed by regression beginning on day 12 and complete disappearance by day 15. Cytotoxic T lymphocytes (CTL) in the spleen and regional lymph nodes were most cytolytic on days 7-9, and this activity decreased slowly during tumor regression. Lytic activity in the blood peaked at day 9 and then decreased rapidly. In cells from the mesenteric nodes, cytolytic activity did not appear until day 14 and peaked at day 21. Cytolytic activity of effector cells within the tumor peaked at day 12 (onset of regression). This activity was abrogated upon treatment with antitheta serum and complement. Spleen cells from mice either bearing a tumor or having recently rejected a tumor were separated by velocity sedimentation at unit gravity. Analysis of the direct cytolytic activity of each cell pool indicated that 94% of the CTL in the spleens of tumor-bearing mice consisted of medium- to large-sized cells. Ninety percent of the precursor cells capable of mounting an enhanced CTL response upon restimulation in mixed lymphocyte culture were small cells. However, spleen cells from day 15 mice (tumor regressed) separated in the same way revealed 82% of active CTL to be among small- to medium-sized cells. Small cells again contained the majority (80%) of the precursors of CTL formed after restimulation in mixed lymphocyte culture. Precursor cells of CTL found after stimulation in allogeneic mixed lymphocyte culture were concentrated in the small and medium-sized lymphocyte fractions obtained from spleens of MSV-treated mice. (12 references)

3911 A COMPARISON OF MEMBRANE GLYCOCONJUGATES FROM MOUSE CELLS TRANSFORMED BY MURINE AND PRIMATE RNA SARCOMA VIRUSES. (Eng.) Fishman, P. H. (Natl. Inst. Neurological and Communicative Disorders and Stroke, Bethesda, MD 20014); Brady, R. O.; Aaronson, S. A. *Biochemistry* 15(1): 201-208; 1976.

Kirsten murine sarcoma virus transformed Balb/3T3 mouse cells (K-Balb) were found to have altered membrane glycoconjugates compared to normal Balb/3T3 cells. There were reduced amounts of mono and disialogangliosides, GM1 and GD1a. Activity of the specific galactosyltransferase required for synthesis of these gangliosides was reduced to between 0 and 18.5% of normal in the several K-Balb clones examined. When fucose-labeled glycopeptides derived from the surfaces of Balb/3T3 and K-Balb cells were compared by gel filtration chromatography, the glycopeptides from the transformed cells were enriched in earlier eluting components. These differences were also observed when the glycopeptides were derived from the entire cell and were diminished when the surface or cellular glycopeptides from Balb/3T3 and K-Balb were digested with neuraminidase prior to chromatographic analysis. The changes in these membrane sialoglycolipids and sialoglycopeptides were not influenced by Rauscher leukemia virus infection. These changes in the membrane glycoconjugates were not observed in Wooley monkey sarcoma virus transformed Balb/3T3 cells (W-Balb). Although W-Balb cells like K-Balb were transformed by tissue culture criteria, their ganglioside composition, galactosyltransferase activity, and glycopeptide patterns were similar to normal Balb/3T3.

3912 RADIATION ACTIVATION OF ENDOGENOUS LEUKEMIA VIRUSES IN CELL CULTURE: ACUTE X-RAY IRRADIATION. (Eng.) Decleve, A.; Niwa, O.; Gelmann, E.; Kaplan, H. S. *In: Biology of Radiation Carcinogenesis*. Edited by J. M. Yuhas; R. W. Tennant; J. D. Regan. (New York: Raven Press): pp. 217-225; 1976.

The radiation activation of endogenous murine lymphoma virus (MuLV) was studied in secondary mouse embryo fibroblasts (BL-MEF) and in a line of established mouse embryo fibroblasts in culture. Initial experiments indicated a small yield of activated virus, as determined by reverse XC and immunofluorescence (IF) assays with direct examination of the target cells. It was concluded that overlaying the irradiated target cells with a detector cell inoculum would have several advantages. Cells were irradiated in suspension with 150-10,000 rads, plated at 2 x 10^5 cells/60-mm dish, and overlaid 1 day later with either BL-5, NIN/3T3, or NRK cells. At 4-day intervals, cultures were subcultured and passed six to eight times. Viral assays were carried out at each passage. In the dose range of 450-10,000 rads, induction was observed in 7/10 experiments. In all positive assays, both of the murine detector cell cultures were positive, but in only one case did NRK cells become positive. There was no difference in the percentage of positive cells between BL-5 and BL-MEF. The phase of the cell culture and the subculture schedule did not influence the virus induction rate, and in synchronous cells there was no increase in viral induction in cells in either S or G_1. The form of the culture, i.e., monolayer or suspension, did not affect viral induction. By varying the target cell number at the time of irradiation, the induction rate was estimated to be 10^{-5}. Induction was observed with BL-5 cells (doubling time = 24 hr) but not with BALB/c-3T3 cells (doubling time = 35 hr). At least 10^2 cells were necessary to serve as efficient detector cells. (21 references)

3913 ON THE NECESSARY AND SUFFICIENT PROPERTIES FOR LEUKEMOGENICITY OF RADIATION LEUKEMIA VIRUSES IN C57BL/6 MICE. (Eng.) Haas, M. (Dept. Cell Biology, Weizmann Inst. Science, Rehovot, Israel). *Bibl Haematol (Basel)* (43): 430-434; 1976.

Studies relating to the loss of leukemogenicity of radiation leukemia virus (RLV) grown in mouse embryo fibroblasts (MEF) are reported. The leukemogenic, lymphoblast line-produced virus infected B6 thymocytes, but not ecotropic or xenotropic cells. MEF-grown RLV infected ecotropic but not xenotropic or thymotropic cells. RLV tumor cells were analyzed for their ecotropic and xenotropic virus content by cocultivation on indicator host cells of various types. B6 thymoma cells induced by x irradiation released only a virus that grew in x-tropic indicator cells and no ecotropic viruses. Most cell-free extracts of these thymomas are inactive in vivo. Cells of an in vivo-enriched, RLV-induced thymoma released both a xenotropic and an ecotropic virus. Cell-free extracts of this thymoma are highly leukemogenic and thymotropic in vivo. Another RLV-induced thymoma line, K127R4, released a virus that grew only in B-tropic indicator host cells. This line produces murine type C viruses that are not leukemogenic in vivo but thymotropic in vitro. Thymoma cells releasing a xenotropic virus only or a B-tropic virus only produced a virus that was nonleukemic in vivo. Cells releasing both x- and B-tropic viruses produced a virus that was highly leukemogenic in vivo. Normal B6 thymocytes released only xenotropic virus (no ecotropic virus). The resistance of spleen cells to infection with RLV might be due to a surface restriction. (19 references)

3914 FINE STRUCTURE OF A MURINE MAMMARY CARCINOMA CELL LINE. (Eng.) Arnold, W. J. (Div. Biological Sciences, Michigan Cancer Foundation, 110 East Warren Ave., Detroit, MI 48201); Soule, H. D.; Russo, J. *In Vitro* 12(1): 57-64; 1976.

A fine structural study was made of cells from the epitheloid MCF-8/5-2A cell line derived from a mouse mammary tumor virus (MuMTV)-free, D2 transplantable hyperplastic outgrowth. Electron microscopy showed the cells to be truly epithelial with many cell-to-cell junctions and microvilli. The cells were similar in many respects to normal mouse mammary gland and some of the conventional mammary tumor-derived cell lines. This study supports previous observations of the absence of MuMTV in MCF-8 within the limits of morphological detection, and demonstrates the presence of numerous virus particles within, or budding into, cisternae of the endoplasmic reticulum and nuclear envelope. This intracisternal A particles have not been previously described in such abundance in mammary tumor tissue culture cells.

3915 RELATIONSHIP BETWEEN RECEPTOR AND MAMMARY TUMOUR VIRUS PRODUCTION AFTER STIMULATION BY GLUCOCORTICOID. (Eng.) Shyamala, G. (Jewish General Hosp., 3755 Cote Ste-Catherine Road, Montreal, Quebec, Canada H3T 1E2); Dickson, C. *Nature* 262(5564): 107-112; 1976.

The presence of cytoplasmic and nuclear glucocorticoid receptors in cultured primary mouse mammary tumor cells is reported, along with the accelerated production of mammary tumor virus (MTV) after 30 min exposure to glucocorticoids and the antagonistic action of progesterone. Binding studies of dexamethasone to receptors in cytoplasmic extracts indicated that saturation was reached at 4×10^{-10} M, and Scatchard plots indicated a single class of binding site. The dissociation constant was estimated to be 1.6×10^{-9}, and the number of binding sites per cell was estimated to be approximately 1,400. Binding activity was destroyed by treatment with pronase or trypsin, but not with DNase or RNase. The order of affinity of steroids for the receptor was dexamethasone > deoxycorticosterone > corticosterone > progesterone > hydrocortisone. Studies of the binding of dexamethasone to nuclear binding sites indicated that specific binding was complete by 30 min and saturation was reached with 10^{-8} M dexamethasone. About 90% of the nuclear bound radioactivity could be solubilized, and the radioactivity was bound to a macromolecule. Pulsing the cell for 30 min was sufficient to evoke a subsequent near maximum stimulation of MTV. There was no evidence for type C virus production. Exposure of cells to progesterone alone (10^{-4} to 10^{-6} M) did not stimulate MTV but neither did it block the basal level of MTV production. At 10^{-5} M progesterone, the stimulation of MTV production by 10^{-6} M dexamethasone was inhibited by 10%, and at 10^{-4} M progesterone, the inhibition was 70%. The failure of progesterone to remain bound to the receptor in the nucleus may be the reason that it cannot stimulate MTV, but its competition with dexamethasone for the cytoplasmic receptor accounts for its antagonism with dexamethasone. (27 references)

3916 AN ANIMAL MODEL FOR DNA-RNA VIRUS INTERACTION BASED UPON VIROLOGICAL AND HISTOLOGICAL FINDINGS. (Eng.) Hsiung, G. D. (Virology Lab., V. A. Hosp., West Haven, CT 06516); McTighe, A. H. *Cancer Res* 36(2): 674-677; 1976.

The expression of the latent herpes virus, generally age and strain dependent as is the development of spontaneous guinea pig leukemia, was investigated. Guinea pigs, particularly older inbred strains including strain 2, showed widespread herpes virus infection. Leukocytes or tissue cells taken directly from infected guinea pigs showed no intracellular viral antigens by immunofluorescence, no intranuclear inclusions by light microscopy, and no virus particles by electron microscopy. The presence of the viral genome was established by demonstrating the presence of virus-induced inclusions and herpesvirus particles following cultivation of the infected tissues. It was found that guinea pig oncornavirus could be induced in all primary cell cultures and passaged cell lines derived from guinea pigs as long as they were exposed to 5-bromo-2'-deoxyuridine (regardless to tissue origin, guinea pig strain or age of the animals.) Studies revealed that, in the presence of the endogenous oncornavirus, a superinfection with herpesvirus led to the development of self-limited lymphoproliferative changes. It is suggested that interaction between the DNA and RNA viruses may play an important role in the natural occurrence of viral oncogenesis.

3917 BIOLOGY OF FELINE LEUKEMIA VIRUS IN THE NATURAL ENVIRONMENT. (Eng.) Hardy, W. D., Jr. (Lab. Veterinary Oncology, Memorial Sloan-Kettering Cancer Center, New York, NY 10012); Hess, P. W.; MacEwan, E. G.; McClelland, A. J.; Zuckerman, E. E.; Essex, M.; Cotter, S. M.; Jarrett, O. *Cancer Res* 36(2): 582-588; 1976.

The environmental control of feline leukemia virus (FeLV) was investigated for feasibility. The observed clustering of cases of feline lymphosarcoma suggested that FeLV was an infectious agent for cats. The development of a simple immunofluorescent test for FeLV permitted a sero-epidemiological study to be undertaken on the distribution of the virus in cats living in their natural environment. Over 2000 cats were tested, and the results showed that FeLV is an infectious agent for cats. After the infectious nature of FeLV was discovered, a simple FeLV test and removal program was devised to control the spread of the virus in the natural environment. The spread of FeLV was controlled in 45 households by removing the FeLV-infected cats, while in 25 households, where the infected cats were left in contact with the uninfected cats, 12% of the uninfected cats became infected. The ultimate control of FeLV, through development of an effective FeLV vaccine, seems feasible since cats have been effectively immunized with attenuated FeLV.

3918 EXPERIMENTAL ONCORNAVIRUSES IN THE CAT. (Eng.) Yohn, D. S. (Dept. Veterinary Pathobiology, Ohio State Univ., Columbus, OH 43210); Olsen, R. G.; Schaller, J. P.; Hoover, E. A.; Mathes, L. E.; Heding, L.; Davis, G. W. *Cancer Res* 36(2): 646-651; 1976.

Feline leukemia virus (FLV) was used to study the control of feline oncornavirus-mediated disease by killed virus vaccines. Immunization of seven pregnant queens over a 6-wk period with UV inactivated Gardner-Arnstein FLV resulted in significant protection among 12 kittens challenged with a tumor-forming dose 90 at 7 days of age. Immunity was not present in kittens challenged at 35 days of age. Among 12 kittens born of queens immunized during pregnancy with Kawakami-Theilen FLV and challenged with the same live virus at 4 days of age, protection was noted, ranging from prolonged survival to complete protection (3 kittens). The higher the antibody titer in the mother, the more effective the protection afforded the kittens. Immunization of 43 kittens during their first 5 wk of life with the same vaccines used in adult cats did not protect against FLV challenge at 5 wk of age. Neutralizing antibody responses in these kittens were significantly lower than in pregnant queens. That kittens of this age are immunologically responsive was established using γ-irradiated (5-7 megarads) crude tumor extract. Nine kittens were completely protected. All kittens developed feline oncornavirus-associated membrane antibodies while three developed demonstrable levels of virus-neutralizing antibodies. It is concluded that killed virus vaccines can control RNA tumor virus oncogenesis in cats and may contribute to the development of such vaccines for humans.

3919 VARIANTS OF SIMIAN VIRUS 40-TRANSFORMED MOUSE CELLS RESISTANT TO CYTOCHALASIN B. (Eng.) Kelly, F.; Sambrook, J. *In: Tumor Viruses. Cold Spring Harbor Symposia on Quantitative Biology.* Cold Spring Harbor Lab. (Cold Spring Harbor, NY): vol. 39, pp. 345-353; 1975.

The ability of cytochalasin B to discriminate normal from transformed cells was used as a basis for selecting from the SVT2 line of SV40-transformed mouse cells a class of variants that shows alterations in the expression of virus-specific functions. Semiconfluent cultures of SVT2 were exposed to cytochalasin B (5 μg/ml), then placed in fresh medium, after which they were placed again in cytochalasin B medium. Two cytochalasin B-resistant sublines, CBR-1 and CBR-2, were among the colonies that resulted. Both lines consisted of small, round, spindly cells that showed the same growth and tumorigenic characteristics as SVT2 but responded differently to cytochalasin B. In the presence of the drug they became binucleated, but only a few cells underwent further nuclear division to become tetranucleated. The SV40 T antigen could not be detected in either of the two resistant lines for 50 or more generations, after which 5%-10% of the cells appeared T-antigen positive. Upon recloning, T antigen-negative cells were obtained. Like the parental STV2 cells, the CBR-1 and CBR-2 cells contained DNA sequences homologous to all four segments of the SV40 genome, but the amounts of fragments A, C, and D were reduced in comparison with SVT2. No infectious virus could be rescued from either resistant line, and the two lines shared the loss of one chromosome 3 (three copies present in SVT2) and one chromosome X (two copies present in SVT2). Each had different marker chromosomes. Studies of this sort will help to appraise the relative contributions of viral and cellular genes to the maintenance of the transformed state. (31 references)

3920 CHARACTERIZATION OF CLONED EVOLUTIONARY VARIANTS OF SIMIAN VIRUS 40. (Eng.) Brockman, W. W.; Lee, T. N. H.; Nathans, D. *In: Tumor Viruses. Cold Spring Harbor Symposia on Quantitative Biology.* Cold Spring Harbor Lab. (Cold Spring Harbor, NY); vol. 39, pp. 119-127; 1975.

The structure of variant simian virus 40 (SV40) genomes is described, and the evolution of SV40 variants and the replication of viral DNA are discussed. Plaques were chosen from both complementation and nonselective cloning procedures and analyzed for SV40 variants. Endonuclease R. *Hind* from *Hemophilus influenzae* endonuclease cleavage and electron microscopic heteroduplex mapping were used to characterize variant genomes. The following observations were made: (a) the paucity of SV40 DNA sequences in variant ev-1103 argues that this segment cannot code for a functional initiator protein, yet it replicates in the presence of helper virus; and (b) in ev-1114 the site where SV40 DNA replication normally terminates is present but is bypassed by the growing fork. They lead to the conclusion that the initiation signal is probably the only cis function involved in SV40 DNA replication. A cis-acting SV40 protein or a specific termination signal is not required. Recombination between SV40 and host DNA cell probably does not occur at a unique host site. Evolutionary SV40 variants are valuable for studying viral and perhaps cellular replication and in defining other SV40 functions such as tumorigenesis. (23 references)

3921 CIRCULAR DICHROISM AND ETHIDIUM BROMIDE BINDING CAPACITY OF CHROMATIN FROM CELLS TEMPERATURE SENSITIVE FOR THE TRANSFORMED PHENOTYPE. (Eng.) Ide, T. (Dept. Pathology, Temple Univ. Sch. Medicine, Philadelphia, PA 19140); Baserga, R. *Biochemistry* 15(3): 600-605; 1976.

A clone of cells (H6-15/163) originally derived from SV-40 transformed 3T3 cells expressed the transformed phenotype at low (32 C) but not at high (39

C) temperature. Chromatin was isolated from these cells grown at either temperature and studied by circular dichroism and for its ability to bind the intercalating dye, ethidium bromide. During the exponential phase of growth the chromatins of cells grown at either 32 or 39 C was undistinguishable. Cessation of growth in confluent cultures produced marked changes in circular dichroism spectra and in ethidium bromide binding capacity of chromatin. The changes were much more pronounced at 39 C (where the cells truly become quiescent) than at 32 C (where cell proliferation continues although the number of cells per culture remains stationary). Temperature shifts and medium replacement also caused changes in chromatin structure, but the changes were related to the extent of cell proliferation. It was concluded that the chromatin changes occurring in 116-15/163 cells and detectable by circular dichroism and ethidium bromide binding were related to the proliferating activity of the cultured cells rather than to the expression of the transformed or untransformed phenotype.

3922 TRANSCRIPTION OF SV40 IN LYTICALLY INFECTED AND TRANSFORMED CELLS. (Eng.) Sambrook, J.; Jackson, A.; Keller, W.; Ozanne, B.; Sharp, P. A.; Sugden, B. *In: Tumor Virus-Host Cell Interaction.* Edited by A. Kolber. (New York: Plenum Press): pp. 91-116; 1975.

At the beginning of simian virus 40 (SV40) DNA replication in monkey cells there is an increase in viral RNA synthesis and a change in transcription. At least some control of virus gene expression is exerted at the level of RNA. All of the early functions of SV40 were shown to be coded by the E or (-) strand of the SV40 DNA (that complementary to cRNA) and the late viral functions by the L or (+) strand. There is at least one strand switch during transcription of late SV40 RNA. To determine the characteristics of SV40-specific transcription, the ability of RNA to hybridize to SV40 DNA was tested. In monkey cells late in SV40 infection, it was host polymerase II that transcribed the viral DNA; the same enzyme was probably responsible for synthesis of both early and late sequences of viral RNA. Early and late regions of the viral genome were mapped. It was assumed that early RNA is transcribed from a contiguous portion of the viral genome. All the early functions of SV40 were located within fragments A and C; all sequences of B and D and part of A and C code for late functions. If all early genes of SV40 are contiguous, strand switches between early and late RNAs occur at positions 55 and 24. Explanations for viral regulation and for the mechanism of the shift from early to late RNA synthesis are offered. Separated strands of SV40 DNA were used to measure the percentage of viral genome transcribed into RNA in mouse SV40 transformants and other cell lines, and the positions of these sequences were mapped. Nearly all transformed cell lines contained RNA complementary to more sequences of E strand DNA than did lytically infected cells. Three premises are used to account for viral RNA synthesis in transformed cells: (a) SV40 DNA integrates into host DNA with a break in its late genes, (b) integration does not necessarily occur at the same site within the viral genome in different cell lines, and (c) integration places viral DNA under the control of host promoter(s). The model indicates that after initiating transcription at a host promoter, the RNA polymerase enters the integrated viral DNA on the E strand in the late region of the genome. It continues along SV40 sequences, synthesizing first ''anti-late'' and then early RNA, before termination occurs at the 3' end of the early genes. (41 references)

3923 GENETICS OF CELL TRANSFORMATION BY SIMIAN VIRUS 40. (Eng.) Croce, C. M.; Huebner, K.; Girardi, A. J.; Koprowski, H. *In: Tumor Viruses. Cold Spring Harbor Symposia on Quantitative Biology.* Cold Spring Harbor Lab. (Cold Spring Harbor, NY): vol. 39, pp. 335-343; 1975.

A series of somatic cell hybrids between mouse cells and simian virus 40 (SV40)-transformed primate cells was studied for the expression of SV40 T antigen and tumor-specific transplantation antigen (TSTA) and for the rescuability of the SV40 genome. The purpose was to determine if the presence of the SV40-induced antigens in the hybrid cells was due to a specific chromosome in which the viral genome is integrated. The results indicated that integration of the SV40 genome into human chromosome 7 results in transformation of the human cells and in the expression of SV40-induced antigens. If integration of the SV40 genome in human chromosomes other than 7 ever occurred, it did not result in cell transformation and the expression of the virus-induced antigens. The expression of the SV40 T antigen in different monkey cells transformed by an adeno 7-SV40 hybrid was also related to a specific monkey chromosome; this chromosome resembles a human group C chromosome. Somatic cell hybrids between normal nondividing mouse cells and SV40-transformed human cells behaved as transformed cells and contained, without exception, the human chromosome 7 carrying the SV40 genome. Since no segregation into SV40 T antigen-negative hybrid clones was observed in these hybrids, it was concluded that the presence of the human chromosome 7 carrying the SV40 genome in the hybrids is mandatory for cell division. The human chromosome 7 apparently contains gene(s) that code for ''transforming factors'' responsible for the transformed phenotype. (33 references)

3924 CLEAVAGE MAP OF THE SIMIAN-VIRUS-40 GENOME BY THE RESTRICTION ENDONUCLEASE III OF *HAEMOPHILUS AEGYPTIUS*. (Eng.) Yang, R. C.-A. (Laboratorium voor Moleculaire Biologie, Fakulteit der Wetenschappen, Rijksuniversiteit te Gent, K.L. Ledeganckstraat 35, B-9000 Gent, Belgium); Van de Voorde, A.; Fiers, W. *Eur J Biochem* 61(1): 101-117; 1976.

Enzymatic digestion of simian virus 40 (SV40) DNA with *Haemophilus aegyptius* restriction endonuclease *Hae* III resulted in 10 major and 8 minor fragments. These were resolved by electrophoresis on graduated polyacrylamide slab gels. All fragments were characterized with respect to the size relative to the *Haemophilus influenzae* Rd fragments (*Hind*). They were ordered on the SV40 DNA map by means of overlap analysis of the double cleavage products derived from sequential digestion of *Hind* fragments with *Hae* III endonuclease and *Hae* fragments with *Hind* II + III enzyme, as well as by other reciprocal cleavage experiments, including those involving *Haemophilus parainfluenzae* fragments. In this way the 18 *Hae* III cleavage sites and the 13 *Hind* sites were localized on the circular SV40 DNA map. The detailed mapping of the cleavage sites by different restriction enzymes on the SV40 genome will provide a reliable basis for the physical location of the various biological functions and for further nucleotide sequence analysis.

3925 EXCISION OF VIRAL DNA FROM HOST CELL DNA AFTER INDUCTION OF SIMIAN VIRUS 40-TRANSFORMED HAMSTER CELLS. (Eng.) Rakusanova, T. (Massachusetts General Hosp., Boston, MA 02114); Kaplan, J. C.; Smales, W. P.; Black, P. H. *J Virol* 19(1): 279-285; 1976.

Viral DNA is covalently linked to cellular DNA in transformed cells. It is demonstrated that the simian virus 40 (SV40) genome is excised from its integrated state prior to viral replication. SV40-transformed inbred hamster kidney cells and sucrose gradient veolcity sedimentation techniques were used to show that the SV40 DNA is linked to cellular DNA by alkali-stable bonds. The Hirt extraction procedure was employed to measure the excision of viral DNA following viral stimulation with either mitomycin C or irradiation. This method follows the movement of DNA from a high molecular wt host cell DNA to a pool of low molecular wt DNA. It was shown that the increase in the low molecular wt DNA fractions is not due to random breakage of cellular DNA. Using irradiation and treatment with cytosine arabinoside, an inhibitor of DNA replication, it was demonstrated that excision of SV40 DNA may have preceded viral DNA replication by approximately 7 hr. (12 references)

3926 INFECTION OF MOUSE BLASTOCYSTS WITH SV40 DNA: NORMAL DEVELOPMENT OF THE INFECTED EMBRYOS AND PERSISTENCE OF SV40-SPECIFIC DNA SEQUENCES IN THE ADULT ANIMALS. (Eng.) Jaenisch, R. *In: Tumor Viruses. Cold Spring Harbor Symposia on Quantitative Biology.* Cold Spring Harbor Lab. (Cold Spring Harbor, NY): vol. 39, pp. 375-380; 1975.

Preimplantation C3H inbred strain mouse blastocysts were infected with purified simian virus 40 (SV40) DNA by microinjection into the blastocoel. The infected embryos developed into mature and apparently healthy mice. Detection and measurement of SV40 sequences in DNA preparations from adult mouse tissues (liver, kidney, and brain) were accomplished by comparison of the reassociation kinetics of highly labeled, denatured SV40 DNA in the presence of DNA from experimental mice, control animals, or calf thymus. Molecular hybridization tests revealed the presence of SV40-specific sequences in some adult tissue. It was suggested that the viral DNA was integrated into the host genome at an early stage of development, but an extrachromosomal existence of the SV40 genome cannot be excluded. The mice were tumor-free at 1 yr of age; tumorigenesis may have required more time to occur. Testing for expression of an integrated viral genome in diverse differentiated tissues may provide a useful model system to study the regulation of differentiation. (19 references)

3927 RANDOM CLEAVAGE OF SUPERHELICAL SV40 DNA BY S_1 NUCLEASE. (Eng.) Waldeck, W. (Institut fuer Virusforschung, Deutsches Krebsforschungzentrum, 69 Heidelberg, West Germany); Chowdhury, K.; Gruss, P.; Sauer, G. *Biochem Biophys Acta* 425(2): 157-167; 1976.

S_1 nuclease-generated SV40 DNA FO III was examined after redigestion with other restriction endonucleases to test the hypothesis that cleavage occurs preferentially, depending on the salt concentrations, at either one of the regions 0.15-0.25 and 0.45-0.55 fractional length from the *Eco*·RI restriction endonuclease cleavage site. Superhelical SV40 DNA FO I was reacted with S_1 nuclease, the reaction product subjected to agarose gel electrophoresis, the section of the gel containing FO III isolated, and the DNA extracted by homogenization. The S_1 FO III was treated with 8 M urea and 0.1 M EDTA and subjected to chromatography on hydroxyapatite to give a reactive FO III that could be subjected to further digestion with restriction endonucleases. S_1 FO III was digested with *Hpa* I and *Hind* III at moderate (50 mM) and high (250 mM) salt concentrations, and the products were electrophoresed in agarose gels. The fragments were electrophoretically indistinguishable from those seen after superhelical SV40 DNA FO I was digested with the corresponding enzyme. When the linear S_1-generated duplexes were melted and renatured, circular duplexes were formed in addition to complex larger structures, indicating that cleavage must have occurred at different sites. The double strand-cleaving activity present in S_1 nuclease preparations required circular DNA as a substrate, as linear SV40 DNA is not cleaved. It is concluded that superhelical SV40 DNA FO I is opened by S_1 nuclease to form linear duplex rods of full length and that cleavage occurs, in contrast to other reports, completely at random and not within one or two regions, regardless of the salt concentrations used. (17 references)

3928 DISSOCIATION AND DNA BINDING OF SV40 T ANTIGEN. (Eng.) Carroll, R. B.; Hager, L.; Dulbecco, R. *In: Tumor Viruses. Cold Spring Harbor Symposia on Quantitative Biology.* Cold Spring Harbor Lab. (Cold Spring Harbor, NY): vol. 39, pp. 291-293; 1975.

By use of salt dissociation, three T antigen species (molecular wt 70,000, 140,000, and 300,000) were isolated from SV3T3 cells. The antigens bound to double-stranded DNA cellulose at pH 6.2. The binding properties were found to be dissimilar to those of phage T4 gene-32 protein but similar to those of the lambda repressor.

3929 ISOLATION OF VIRAL SPECIFIC RNA FROM SV40 INFECTED CELLS BY VIRAL DNA CHEMICALLY LINKED TO A CELLULOSE MATRIX. (Eng.) Shih, T. Y. (Natl. Cancer Inst., Natl. Inst. Health, Bethesda, MD 20014); Khoury, G. *Biochemistry* 15(3): 487-493; 1976.

Simian virus 40 (SV40) DNA fragments chemically attached to neutral cellulose powder with a water-soluble carbodiimide were used to isolate late lytic viral specific RNA from virus infected cells. Cytoplasmic RNA was extracted from BSC-1 cells 48 hr post-infection with SV40 at a multiplicity of 20-40 PFU/cell. Exhaustive hybridization to SV40 DNA revealed that virtually all of the isolated RNA molecules contain SV40 specific sequences. Comparison with SV40 cRNA prepared with purified *Escherichia coli* RNA polymerase and a SV40 DNA I template suggested that the purity of the isolated SV40 specific RNA is very close to 100%. The background level for the nonspecific binding of RNA to a purified cellulose matrix is very low. Retention of nonspecific RNA by SV40 DNA-cellulose is only 1.5% of the viral specific RNA isolated under saturating conditions for the column. Sedimentation in neutral sucrose suggests that the major 16S viral specific RNA was isolated largely intact.

3930 ''EARLY'' SIMIAN-VIRUS-40-SPECIFIC RNA CONTAINS INFORMATION FOR TUMOR ANTIGEN FORMATION AND CHROMATIN REPLICATION. (Eng.) Graessmann, M. (Institut fuer Mulekularbiologie und Biochemie der Freien Universitat Berlin, West Berlin, 33 Arnimallee 22, Germany); Graessmann, A. *Proc Natl Acad Sci USA* 73(2): 366-370; 1976.

The temporal and quantitative relation between the synthesis of the early virus-specific RNA and the subsequent sequence of events was investigated. Simian virus 40 (SV40)-specific RNA, complementary to the early viral DNA strand, was synthesized in vitro by Escherichia coli DNA-dependent RNA polymerase. Following thorough removal of the SV40 DNA by exhaustive DNase treatment and equilibrium density centrifugation in Cs_2SO_4 the cRNA preparation was injected into epitheloid cells of confluent primary mouse culture by a microinjection technique and cRNA-induced T-antigen formation and DNA synthesis were investigated. SV40-induced tumor (T)-antigen formation, chromatin replication, and mitosis in primary mouse kidney cells arrested in G_0 phase of the mitotic cycle. Results supported the hypothesis that T-antigen is a virus-coded protein and that the early virus-specific mRNA contains information necessary for stimulation of cellular DNA replication in the arrested cells.

3931 TEMPERATURE-DEPENDENT SYNTHESIS OF EARLY AND LATE SV40 CYTOPLASMIC RNA DURING *tsD*101* MUTANT VIRION INFECTION. (Eng.) Robb, J. A. (Dept. Pathology, Sch. Medicine, Univ. California, San Diego, La Jolla, CA 92093); Lopez-Revilla, R. *Intervirology* 6: 122-128; 1975/76.

Hybridization techniques were used to study the transcription of RNA from temperature-sensitive (*ts*) viruses at restrictive temperatures. TC7 cells were infected with the D group *ts* mutants of simian virus 40 (SV40) papovavirus B2, represented by *tsD*101* (D101), and early and late transcription was followed with the use of radioactive viral RNA. Since (a) the majority of SV40-specific RNA is transcribed as late RNA and (b) transcription of late RNA requires SV40 DNA replication, cytosine arabinonucleoside (Ara-C, an inhibitor of DNA synthesis) was used to distinguish between early and late transcription. The amount of SV40-specific RNA synthesized by D101 virus-infected cells at restrictive temperatures in the presence of Ara-C was reduced twentyfold compared to that synthesized at permissive temperatures. The small amount of viral RNA synthesized can be accounted for by the ''leakiness'' of the mutant phenotype. It is concluded that all transcription of the D group genome is inhibited at restrictive temperatures during infection. The inhibition is probably due to an inability of the infecting mutant virion to be sufficiently uncoated within the nucleus. This leads to inhibition of the cell-directed synthesis of the viral D protein required for early transcription. (22 references)

3932 ISOLATION AND CHARACTERIZATION OF INDIVIDUAL CLONES OF SIMIAN VIRUS 40 MUTANTS CONTAINING DELETIONS, DUPLICATIONS, AND INSERTIONS IN THEIR DNA. (Eng.) Mertz, J. E.; Carbon, J.; Herzberg, M.; Davis, R. W.; Berg, P. *In: Tumor Viruses. Cold Spring Harbor Symposia on Quantitative Biology.* Cold Spring Harbor Lab. (Cold Spring Harbor, NY): vol. 39, pp. 69-84; 1975.

Various methods used for the generation, isolation, propagation, and characterization of deletion and insertion mutants of simian virus 40 (SV40) are summarized. The following approaches were used: (a) serial passaging of SV40 at high multiplicities; (b) isolation and propagation of individual clones of naturally arising defective deletion mutants by complementation with helper virus; (c) construction of deletion mutants from wild-type SV40 DNA with *Escherichia coli* restriction endonuclease; (d) selective isolation of a class of viable deletion mutants using *Hemophilus parainfluenzae* restriction endonuclease; (e) construction of insertion mutants in vitro; and (f) the study of mutants generated by cell-mediated ring closure of exonuclease-treated linear SV40 DNA. Mutants of these types should be useful for genetic analysis of SV40. (49 references)

3933 THE SEMIAUTONOMOUS REPLICON: A MOLECULAR MODEL FOR THE ONCOGENICITY OF SV40. (Eng.) Martin, R. G.; Chou, J. Y.; Avila, J.; Saral, R. *In: Tumor Viruses. Cold Spring Harbor Symposia on Quantitative Biology.* Cold Spring Harbor Lab. (Cold Spring Harbor, NY); vol. 39, pp. 17-24; 1975.

A large number of temperature-sensitive (ts) mutants of simian virus 40 (SV40) were isolated and characterized. Their behavior in permissive cells and studies of transformation and of the transformed cell lines induced by the mutants are summarized. A model is presented to account for the oncogenicity of SV40. Sixty-eight ts SV40 mutants were classified into five groups (A, B, C, BC, and D) on the basis of their behavior in complementation tests, and their transformation properties were characterized with use of secondary adult Chinese hamster lung cells. It was suggested that: (a) the expression of A function by an integrated SV40 genome in a nonpermissive or semipermissive cell line leads to aberrant initiation of host DNA synthesis; (b) that the majority of parameters generally associated with transformation are normal properties of cells in the S, G_2, or M phase of growth; and (c) that the expression of A function is determined by its site of integration and therefore is not fully autonomous. This model can explain different levels of SV40 transformation, but only if a hierarchy of phenotypes exists. The expression of the viral initiator would determine how long a cell stays in G_0, and this, in turn, would determine to what extent ''transformation'' properties are manifest. (38 references)

3934 STRUCTURAL STUDIES ON TWO ADENOVIRUS 2-SV40 HYBRIDS CONTAINING THE ENTIRE SV40 GENOME. (Eng.) Kelly, T. J., Jr.; Lewis, A. M., Jr.; Levine, A. S.; Siegel, S. *In: Tumor Viruses. Cold Spring Harbor Symposia on Quantitative Biology*. Cold Spring Harbor Lab. (Cold Spring Harbor, NY); vol. 39, pp. 409-417; 1975.

Two adenovirus 2 (Ad2)-simian virus 40 (SV40) hybrids (designated Ad2^{++}HEY and Ad2^{++}LEY) were studied. which Ad2^{++}LEY and Ad2^{++}HEY yield SV40 after infection the length of the repetitious segment. Ad2^{++}LEY and Ad2^{++}HEY may serve as useful models for studying the of the Ad2 genome; the polarity of the inserted SV40 DNA is opposite in the two cases; and the end points of the integrated SV40 DNA segments map at different positions in the physical map of SV40. In spite of their differences, the hybrids within each family are closely related and are probably interconvertible by genetic recombination. The data reported offer an explanation for the difference in efficiency with which Ad2LEY and Ad2HEY yield SV40 after infection of monkey cells. The length of the terminal repetition in LEY-II molecules is on the order of 250 nucleotide pairs, whereas that in HEY-II is over 2,000 pairs and that in HEY-III is over 7,000 pairs. The probability of excision of an intact SV40 genome (perhaps a necessary prerequisite to infectious SV40 virion production) would possibly be a function of the length of the repetitious segment. Ad2LEY and Ad2HEY may serve as useful models for studying the properties of integrated SV40 genomes. (25 references)

3935 STATE OF THE VIRAL DNA IN RAT CELLS TRANSFORMED BY POLYOMA VIRUS. I. VIRUS RESCUE AND THE PRESENCE OF NONINTEGRATED VIRAL DNA MOLECULES. (Eng.) Prasad, I. (New York Univ. Sch. Medicine, New York, NY 10016); Zouzias, D.; Basilico, C. *J Virol* 18(2): 436-444; 1976.

When polyoma virus interacted with a continuous line of rat cells, the infection did not cause virus multiplication but induced transformation. Transformed cells did not produce infectious virus, although in all clones tested virus could be rescued upon fusion with permissive mouse cells. Transformed rat cells contained, in addition to integrated viral genomes, 20 to 50 copies of nonintegrated viral DNA equivalents per cell (average). Nonintegrated viral DNA molecules were also found in cells transformed by the ts-a and ts-8 polyoma mutants and maintained at 33 C. These ''free'' viral DNA particles were not due to a virus carrier state, because the number of nonintegrated viral DNA molecules was unchanged when cells were grown in antipolyoma serum. Recloning of the transformed cell lines produced subclones, which also contained free viral DNA. Most of these molecules were supercoiled and were found in the nuclei of the transformed cells. The nonintegrated viral DNA is infectious; that its specific infectivity is about 100-fold lower than that of polyoma DNA extracted from productively infected cells suggests that these molecules contain a large proportion of defectives. This free viral DNA may have originated from an integrated template.

3936 POLYOMA VIRUS--A STUDY OF WILD-TYPE, MUTANT AND DEFECTIVE DNAs. (Eng.) Fried, M.; Griffin, B. E.; Lund, E.; Robberson, D. L. *In: Tumor Viruses. Cold Spring Harbor Symposia on Quantitative Biology*. Cold Spring Harbor Lab. (Cold Spring Harbor, NY): vol. 39, pp. 45-52; 1975.

The restriction enzyme fragments of the polyoma wild-type strain A-2 and the restriction enzyme cleavage patterns of different temperature-sensitive (ts) mutants and purified defective DNA isolates are analyzed. A physical map of polyoma DNA is described. Preliminary experiments indicate that the transforming activity of polyoma DNA is located in the *Hemophilus influenzae* endonuclease III-55% fragment. The *H. parainfluenzae* endonuclease II cleavage patterns for a number of ts mutants were tabulated. Various defective molecules, which result from the passage of polyoma virus at high multiplicity, have been cloned in the presence of infectious helper molecules. Defective DNA from these clonal isolates has been purified; it is resistant to *Escherichia coli* restriction enzyme I cleavage and interferes with the production of infectious virus. Further analysis of three defective molecules (D-92, D-74, and D-50) with restriction enzymes allowed the construction of physical maps for the D-92 and D-50 species. The defective molecules may be useful in defining the wild-type genome. (11 references)

3937 BIOGENESIS AND CHARACTERIZATION OF SV40 AND POLYOMA RNAS IN PRODUCTIVELY INFECTED CELLS. (Eng.) Aloni, Y. *In: Tumor Viruses. Cold Spring Harbor Symposia on Quantitative Biology*. Cold Spring Harbor Lab. (Cold Spring Harbor, NY); vol. 39, pp. 165-178; 1975.

The mechanism of the shift from early to late RNA synthesis in simian virus 40 (SV40)-infected monkey cells (BSC-1) was investigated. Evidence was accumulated concerning the existence of a control mechanism late after infection in productively infected cells. Asymmetric viral RNA, which contains all the sequences equivalent to one SV40 DNA strand, is protected from RNase digestion by self-annealing with denatured double-stranded RNA purified from infected cells. The length of the viral double-stranded RNA, as measured by electron microscopy, is the same as that of the SV40 DNA molecule. Precursor viral RNA molecules with a molecular wt comparable to a complete transcript of the SV40 genome exhibited a high degree of self-complementarity. It was suggested that the symmetric RNA transcripts are then processed in the nucleus. The final RNA products sediment in sucrose gradients at about 19S; this peak seems to be composed of RNA transcribed from both the early and late regions of the SV40 DNA molecule. It was further suggested that the nuclear 19S components are then transported to the cytoplasm, where some of them are processed to 16S viral RNA; this is the predominant viral RNA component accumulating in the cytoplasm of the infected cells. These experiments indicate that late in the infection of cells, SV40 DNA is transcribed symmetrically over its entire length. (48 references)

3938 POLYOMA DNA REPLICATION: LOCATION OF THE ORIGIN IN DIFFERENT VIRUS STRAINS. (Eng.)

Crawford, L. V.; Robbins, A. K.; Nicklin, P. M.; Osborn, K. *In: Tumor Viruses. Cold Spring Harbor Symposia on Quantitative Biology.* Cold Spring Harbor Lab. (Cold Spring Harbor, NY): Vol. 39, pp. 219-225; 1975.

Restriction enzyme digestion techniques were employed to map and determine the origin of replication of DNA from various mutants of DLP 1106 and Toronto large-plaque strains of polyoma virus. Two estimates of the origin of the DNA replication were arrived at independently. The first located the origin at 29% ± 2% from the *Eco*RI site measured either clockwise or counterclockwise on the DNA map. This estimate assumes that replication proceeds at equal rates in both directions from the origin and that the origin is halfway between the measured positions of the two replication forks. The kinetics of labeling the *Hap*II fragments suggest that the origin exists between 23.2%-29.4% from the *Eco*RI site. These independent estimates agree with each other and with those made by others using electron microscopic techniques. The authors discuss the complications involved in their estimates and compare the DNA maps for the polyoma virus with that of SV40 virus.

3939 PHENOMENA OF BLOOD-BRAIN IMMUNOLOGICAL BARRIER IN THE POLYOMA VIRUS-SYRIAN HAMSTER SYSTEM. (Fre.)

Pagis-de Micco, C. (Unite 119 de l'I.N.S.E.R.M. et Laboratoire de Neuropathologie de la Faculte de Medecine de Marseille, 27, boulevard Jean-Moulin, 13385 Marseille Cedex 4, France); Tripier, M.-F.; Hassoun, J.; Toga, M. *C R Acad Sci [D] (Paris)* 282(7): 683-685; 1976.

The penetration of the blood-brain barrier (BBB) by factors of the virus-induced, antitumoral immune reaction is appraised. The degree of protection obtained by sc and ic injections of cells transformed by the same virus is studied. The experiment involved 60 golden Syrian hamsters. Three injections of 10^7 plaque-forming units of virus, five days apart, were given sc to 20 animals, ic to 20 others. The 20 remaining rodents served as controls. Three groups of 10 hamsters (some protected sc, some protected ic, some controls) received 10^5 tumor cells sc; three other groups of 10 received 10^4 cells ic. The animals with sc protection developed a smaller number of skin and brain tumors (1/10 in each case) than the unprotected control group (10/10 and 7/10). The difference between the animals with cerebral protection and controls was insignificant for both types of tumors. The delays before the appearance of tumors were longer in the animals with effective protection (21 to 28 days) than in the unprotected or less-protected animals (8 to 18 days). The sc injections prevented the induction of sc tumors as well as that of ic tumors. This tended to prove the permeability of the BBB to the efferent flow of the immune reaction. The ic injections did not protect the hamsters against the induction of sc tumors. The antigenic information introduced into the CNS apparently remained secluded within the barrier. The following explanation is proposed: the CNS holds all the elements required for an immune reaction, but the BBB may prevent the release of these elements out of the nervous system while not opposing their penetration.

3940 VIRUS-SPECIFIC RNA IN CELLS PRODUCTIVELY INFECTED OR TRANSFORMED BY POLYOMA VIRUS.

(Eng.) Kamen, R.; Lindstrom, D. M.; Shure, H.; Old, R. W. *In: Tumor Viruses. Cold Spring Harbor Symposia on Quantitative Biology.* Cold Spring Harbor Lab. (Cold Spring Harbor, NY): vol. 39, pp. 187-198; 1975.

Methods used in the study of simian virus 40 (SV40) transcription were applied to the analysis of polyoma-specific RNA sequences present in productively infected transformed cells. Virus-specific RNA found in 3T6 cells at two stages of polyoma infection is characterized. All of the experiments involved hybridization of ^{32}P-labeled viral DNA to nonradioactive RNA extracted from infected cells. A model for polyoma transcription during productive infection is suggested in which the two strands of polyoma DNA are transcribed with different frequencies at various times of infection; however, RNA chain termination is inefficient. A symmetric ''promoter'' region in the vicinity of the *Haemophilus parainfluenzae* endonuclease II-5 (HpaII-5)/HpaII-3 junction is postulated. Prior to DNA replication, transcription is most often initiated on the E strand and proceeds around the genome without specific termination. Processing nucleases would clip out the appropriate mRNA sequences, which would then receive 3'-terminal polyadenylic acid and be transported into the cytoplasm. The onset of viral DNA replication, which initiates at a point very close to the map position of the 5' ends of the polyoma mRNA's, would switch the promoter to favor more frequent initiation on the DNA L strand. Due to a lack of an efficient termination mechanism, the entire L strand would be transcribed. Another possible model that places all control at the level of posttranscriptional modification is also described. Symmetric transcription followed by messenger selection through RNA processing may be a common property of tumor viruses. (41 references)

3941 ENDONUCLEOLYTIC CLEAVAGE OF POLYOMA VIRUS DNA: GENERAL PROPERTIES AND SITE SPECIFICITY OF THE VIRION-ASSOCIATED ENDONUCLEASE. (Eng.) Parodi, A.; Rouget, P.; Croissant, O.; Blangy, D.; Cuzin, F. *In: Tumor Viruses. Cold Spring Harbor Symposia on Quantitative Biology.* Cold Spring Harbor Lab. (Cold Spring Harbor, NY): vol. 39, pp. 247-254; 1975.

The properties and site specificity of a specific endonuclease associated with polyoma virus were studied. Bivalent cations at an optimal concentration of about 5 mM are required for the activity, the optimal pH lies between 7 and 8.5, and the apparent Km on SV40 or polyoma form I DNA is 3 μg/ml. Cleavage rates were about equal on superhelical and single-stranded DNA, and the reaction rate on nonsuperhelical, covalently closed DNA was about five times lower than on form I DNA. The product of the virus-associated nuclease is a unit-length linear molecule with no detectable cohesive ends; a maximum number of one phosphodiester cleavage is made per DNA strand. The enzyme cleaves polyoma DNA form I at a unique site and SV40 DNA at one of two possible sites. In both cases, the cleavage sites appear to coincide with A-T-rich regions, which are partially melted in superhelical molecules. The enzyme appears to be analogous in its type of specificity to Aspergillus endonuclease S_1. The enzyme might recognize only certain site(s) on denatured DNA, and the reaction observed on relaxed double-stranded DNA, or at least the first single-strand break, could be site-specific. It is unknown whether the enzyme recognizes only regions of partial denaturation or whether it requires a more strictly defined structure (either a nucleotide sequence or a peculiar secondary structure). The enzyme could be part of a site-specific recombination mechanism, which might also be involved in integration. (20 references)

3942 PARTIAL PURIFICATION AND PROPERTIES OF A DNA-BINDING PROTEIN FROM NUCLEI OF CELLS INFECTED WITH POLYOMA VIRUS. (Eng.) Yeh, W.-S. (Div. Biology, Kansas State Univ., Manhattan, KS 66506); McGuire, M.; Center, M. S.; Consigli, R. A. *Biochim Biophys Acta* 418(3): 266-276; 1976.

The partial purification and properties of a DNA-binding protein from polyoma-infected 3T3 cell nuclei are described. The assay used to detect this activity measured the amount of double-stranded DNA retained on a nitrocellulose membrane filter in the presence of binding protein. The interaction between DNA and protein was salt-dependent and occurred optimally at 0.8 M NaCl. The isolated protein could bind to both circular and linear duplex DNA. Incubation of the binding protein with PM2 or polyoma DNA resulted in the formation of a fast sedimenting DNA structure in neutral sucrose gradients. The isolated binding protein was also capable of producing a considerable stimulation of both *Escherichia coli* (Pol I) and T4 DNA polymerase activities when either single-stranded or intact, native T7 DNA was used as the template. The binding protein itself was free of detectable DNA polymerase or nuclease activity. These results suggest that the stimulatory effect of the binding protein on the polymerases is not due to the presence of nuclease activity which creates a template primer DNA structure.

3943 INDIRECT COMPLEMENTATION OF A NONTRANSFORMING MUTANT OF POLYOMA VIRUS. (Eng.) Benjamin, T. L.; Goldman, E. *In: Tumor Viruses. Cold Spring Harbor Symposia on Quantitative Biology.* Cold Spring Harbor Lab. (Cold Spring Harbor, NY): vol. 39, pp. 41-44; 1975.

To distinguish between direct and indirect complementation, an extended host range analysis of nontransforming polyoma host range mutant NG18 was undertaken. The results indicated that several categories of nonpolyoma transformed cells permit the growth of NG18. Essentially the same results were obtained with use of three other independently isolated host range mutants similar to NG18. These results verify the indirect complementation hypothesis and rule out direct complementation as a necessary mechanism. This suggests that the polyoma virus requires a certain cellular function(s) for growth, and loss of this ability leads to inability to grow in cells not expressing the required function(s).

3944 VIRION PROTEINS OF POLYOMA TEMPERATURE-SENSITIVE MUTANTS: LATE MUTANTS. (Eng.) Friedmann, T.; Eckhart, W. *In: Tumor Viruses. Cold Spring Harbor Symposia on Quantitative Biology.* Cold Spring Harbor Lab. (Cold Spring Harbor, NY): vol. 39, pp. 243-246; 1975.

Characterization of strains ts 441 and ts 610 of polyoma virus indicated that they were complementary to a third mutant, ts 1260. Further studies suggested that the mutations in ts 441 and ts 610 affected the capsid proteins and that these were responsible for the temperature sensitive defect. The mutation in ts 441 probably caused the replacement of a similarly charged but slightly smaller amino acid. The change in ts 610 was more complicated due to the possible overlap of a major and minor polypeptide. These two mutants are complementary to ts 1260 possibly because the mutation in ts 1260 codes for a different protein than the mutations in the other two or because the mutation codes for a major capsid protein that exhibits intracistronic complementation with ts 441 and ts 610.

3945 INTERMOLECULAR DISULFIDE BONDS: AN IMPORTANT STRUCTURAL FEATURE OF THE POLYOMA VIRUS CAPSID. (Eng.) Walter, G.; Deppert, W. *In: Tumor Viruses. Cold Spring Harbor Symposia on Quantitative Biology.* Cold Spring Harbor Lab. (Cold Spring Harbor, NY): vol. 39, pp. 255-257; 1975.

To investigate the structure of polyoma virus and study the physical and chemical interactions between different virion proteins, sodium dodecyl sulfate and dithiothreitol were used as probes. The results indicated that the capsid represents a stable, highly polymeric structure of VP1 subunits that are covalently linked by disulfide bridges. VP2 and VP3 are bound to this structure by hydrogen bonds. A 100S particle, an intermediate in sodium dodecyl sulfate degradation, contained the polyoma DNA forms I and II in the same ratio and with the same density as untreated virions. It also contained all the viral protein but a reduced amount of histones and was at least 100 times less infectious than the undegraded virus. VP1 was identified as the polyoma virus agglutinin.

3946 CYTOSINE ARABINOSIDE- AND INTERFERON-MEDIATED CONTROL OF POLYOMA AND SV40 GENOME EXPRESSION. (Eng.) Brandner, G.; Mueller, N. *In: Tumor Viruses. Cold Spring Harbor Symposia on Quantitative Biology.* Cold Spring Harbor Lab. (Cold Spring Harbor, NY): vol. 39, pp. 305-308; 1975.

Studies on African green monkey kidney cells infected with polyoma and SV40 (777) indicated that SV40 transcription is primarily affected by interferon. However, interferon and cytosine arabinoside also inhibited the host DNA replication. This interruption has no effect on the current transcription of the late viral genes, indicating that the onset of viral DNA replication is a prerequisite for the persistence of the late SV40 and polyoma genome transcription. Pretreatment with poly(I):poly(C) almost completely inhibited SV40 T antigen formation, suggesting that this control is a primary action of interference on SV40 translation.

3947 FINGERPRINTS OF POLYOMA VIRUS PROTEINS AND MOUSE HISTONES. (Eng.) Fey, G.; Hirt, B. *In: Tumor Viruses. Cold Spring Harbor Symposia on Quantitative Biology.* Cold Spring Harbor Lab. (Cold Spring Harbor, NY): vol. 39, pp. 235-241; 1976.

The structural proteins of highly purified polyoma virus were examined by two-dimensional fingerprint analysis of their tryptic digests. The peptides were reacted on thin-layer sheets with fluorescamine and detected by fluorescence under UV light. Only the VP1 capsid protein and the dominant species of the VP2 and VP3 triplets were studied. Approximately 30 diagnostic spots were detected on the two-dimensional fingerprint of VP1. Fourteen major spots were found for both VP2 and VP3, 10 of which were common to both proteins. It appears that VP2 and VP3 contain significant proportion of common amino acid sequences. The tryptic peptides of VP1 differed from those of VP2 and VP3; only two spots were common to all three fingerprints. A similar comparison between VP1 and VP1a indicated that the two are related. Histones prepared from the nuclei of primary mouse kidney cells were eluted from 2.5 M urea-polyacrylamide gels and from SDS-polyacrylamide gels. Virus core polypeptides were eluted from SDS-polyacrylamide gels only. The results suggested that histones f3, f2b, f2a2, and f2a1 are present in the virus, whereas f1 is absent, and that the proteins eluted from the f3 and f2b bands were mutually cross-contaminated, whereas f2a2 and f2a1 were pure. It was concluded that the virus proteins comigrating with host histones upon SDS- and acid-urea gel electrophoresis contain predominantly the same tryptic peptides as mammalian histones. A similar study of virus-infected cells indicated that the host histone material synthesized prior to infection was integrated into virus particles as well as some histone material synthesized after infection. (18 references)

3948 SYNTHESIS OF THE ADENOVIRUS-CODED DNA BINDING PROTEIN IN INFECTED CELLS. (Eng.) Gilead, Z. (St. Louis Univ. Sch. Medicine, St. Louis, MO 63110); Sugawara, K.; Shanmugam, G.; Green, M. *J Virol* 18(2): 454-460; 1976.

Synthesis of the 75K (molecular wt 70,000-75,000) DNA binding protein, an early virus-coded protein in adenovirus 2-infected KB cells, and its regulation were studied by radioimmune precipitation inhibition assay. The protein was first detected 4 hr postinfection and accumulated at an exponential rate. An arrest of further synthesis (accumulation) was observed at 10 to 11 hr postinfection, coinciding with the synthesis of late virion proteins. In contrast, when the infected cells were treated with arabinosyl cytosine (25 μg/ml) to block viral DNA replication, the synthesis of this protein did not cease but continued for up to 36 hr postinfection. The synthesis of 75K protein in cells, after release from a cycloheximide block (2 to 9 hr postinfection) was analyzed. Although increased amounts of early adenovirus-specific mRNA generally accumulate in infected cells during such a block, treatment did not produce increased levels of this protein, and an abrupt arrest of its formation again occurred upon the synthesis of late virion proteins. Partition of the 75K protein between the nuclear and cytoplasmic fractions during the infection was studied. The protein appeared first in the cytoplasm and then in the nucleus after a slight lag. Accumulation of the protein continued both in the cytoplasm and nucleus, with higher levels being found in the cytoplasm.

3949 EXTENT OF TERMINAL REPETITION IN ADENOVIRUS 2 DNA. (Eng.) Arrand, J. R.; Keller, W.; Roberts, R. J. *In: Tumor Viruses. Cold Spring Harbor Symposia on Quantitative Biology.* Cold Spring Harbor Lab. (Cold Spring Harbor, NY); vol. 39, pp. 401-407; 1975.

Specific endonucleases were used to obtain a more exact estimate of the length of the terminal repetition in adenovirus 2 (Ad2) DNA. The analysis was based on the fact that within an inverted terminal repetition, recognition and cleavage of the DNA by a specific endonuclease form identical terminal fragments, whereas cleavage outside the repetition yields dissimilar fragments from each end. The observations suggested that the inverted terminal repetition is between 100 and 140 nucleotide pairs long. This general approach has wide applications: (a) by careful choice of specific endonucleases any desired region of a DNA molecule may be selected for direct sequence analysis; (b) exonuclease III and reverse transcriptase can be used to label any linear DNA in vitro to a very high specific activity; and (c) by labeling after various extents of exonuclease III digestion and then cleaving with a specific endonuclease, it is possible to map the order of fragments by correlating the appearance of a label in a fragment with extent of exonuclease III digestion.

3950 RNA TRANSCRIPTION IN CULTURES PRODUCTIVELY INFECTED WITH ADENOVIRUS 2. (Eng.) Craig, E. A.; Tal, J.; Nishimoto, T.; Zimmer, S.; McGrogan, M.; Raskas, H. J. *In: Tumor Viruses. Cold Spring Harbor Symposia on Quantitative Biology*. Cold Spring Harbor Lab. (Cold Spring Harbor NY); vol. 39, pp. 483-493; 1975.

The six unique fragments of the viral adenovirus type 2 (Ad2) genome generated by digestion with the restriction endonuclease I from *Escherichia coli* (*Eco*R·RI) were used to study RNA transcription in infected cultures. Although total polyadenylic acid-containing RNA labeled at late times hybridized to B fragment DNA, discrete viral RNAs transcribed from the B fragment at 18 hr were not identified; the late cytoplasmic RNAs transcribed from B may be different from those synthesized early. At early times there was no hybridization of F fragment DNA with cytoplasmic RNA, and late cytoplasmic RNA formed hybrids with 33% of F fragment DNA (corresponding to 0.36×10^6 daltons of RNA). The F-specific sequences are insufficient to code for a 23S molecule and transcription must continue into B or D fragments. No mRNAs transcribed from the D fragment at late times were identified; the D fragment mRNAs transcribed at late times are probably early sequences. The liquid phase hybridizations with early cytoplasmic RNA confirm that there cannot be an in vivo 26S early RNA transcribed solely from C fragment DNA. The block in virus replication was characterized in temperature-sensitive (ts) cell mutants. T23 and T22 might not allow virus adsorption or uncoating at 39 C. Upon a shift to 38 C, ts AF8 cells are blocked in the G1 phase of the cell cycle so that DNA synthesis does not begin. With the appropriate cell mutants, several aspects of host-virus interaction could be clarified. (34 references)

3951 AN EXAMINATION OF THE NUCLEAR RNA OF ADENOVIRUS-TRANSFORMED CELLS. (Eng.) Georgieff, M.; Bachenheimer, S.; Darnell, J. E. *In: Tumor Viruses. Cold Spring Harbor Symposia on Quantitative Biology*. Cold Spring Harbor Lab. (Cold Spring Harbor, NY): vol. 39, pp. 475-482; 1975.

The location within heterogenous nuclear RNA (HnRNA from virus-transformed cells) of virus-specific sequences apparently destined to function as mRNA in the cytoplasm is described. Additional substantiating evidence that HnRNA in general is a precursor to mRNA is reviewed. The use of polyadenylic acid [poly(A)] as a marker appears to be valid. Large, nuclear, poly(A)-terminated molecules containing virus-specific (adenovirus 2) RNA were identified; the great majority of these sequences were actually a 20S species that sedimented similarly to the major 20S cytoplasmic Ad2 RNA. If the biosynthetic pathway of the 20S Ad2 RNA is first as part of a long molecule that is then processed, then a very small part (perhaps 5% or less) of the total poly(A)-containing nuclear RNA represents unprocessed HnRNA transcripts. (34 references)

3952 CLEAVAGE MAPS OF DNA FROM ADENOVIRUS TYPES 2 AND 5 BY RESTRICTION ENDONUCLEASES *Eco*RI AND *Hpa*I. (Eng.) Mulder, C.; Arrand, J. R.; Delius, H.; Keller, W.; Pettersson, U.; Roberts, R. J.; Sharp, P. A. *In: Tumor Viruses. Cold Spring Harbor Symposia on Quantitative Biology*. Cold Spring Harbor Lab. (Cold Spring Harbor, NY): vol. 39, pp. 397-400; 1975.

The fragments of adenovirus types 2 and 5, produced by the endonucleases R·*Eco*RI and R·*Hpa*I, were mapped by a combination of methods: partial denaturation mapping, cleavage of partial digest products, cleavage of specific fragments with a different endonuclease R, and cleavage of end-labeled DNA molecules. The results indicated that the fragments of adenovirus type 2 were arranged ABFDEC and those of type 5 were arranged ACB.

3953 EFFECT OF GONADECTOMY AND EXOGENOUSLY ADMINISTERED SEX HORMONES ON ONCOGENESIS INDUCED BY BOVINE ADENOVIRUS TYPE 3. (Bul.) Silianovska, K. (MA, Oncological Center, Bulgaria). *Onkologia* 12(2): 53-57; 1975.

Research was conducted to determine the effect of gonadectomy and exogenously administered sex hormones on the oncogenesis induced by the bovine WBR adenovirus type 3. A total of 144 Syrian JCV hamsters were injected with virus material at birth and divided in six groups. Two groups of male and female hamsters underwent gonadectomy only; two groups went through the same procedure and were treated with estradiol or testosterone once a month. Two were the control groups, which were injected with virus material only. Bovine adenovirus type 3 serum, kept in the initial cell culture of calf kidney with the titer 10^3 TCD_{50}/0.1 ml, was used in the experiment. The hamsters were inoculated sc with 0.2 ml of the virus within 24 hr after birth. They were subjected to gonadectomy 20-25 days later. A week later, two groups of hamsters began to receive 0.25 mg of estradiol or 1.25 mg of testosterone once a month. The animals were observed for 240 days. It is concluded that the gonadectomy performed on the hamsters of either sex essentially reduced the incidence of neoplasms and prolonged their latent period. Combined castration and exogenous administration of hormone increased tumor incidence in male animals to the level of female controls.

3954 ON THE ULTRASTRUCTURE OF TUMOURS INDUCED WITH BOVINE ADENOVIRUS TYPE 3 IN HAMSTERS. (Eng.) Dobreva, A. M. (Medical Acad., Sofia, Bulgaria); Meshkoy, T. S.; Silyanovska, K. K. *C R Acad Bulg Sci* 28(12): 1709-1712; 1975.

Tumors induced with bovine adenovirus type 3 (BAV 3) in Syrian hamsters were examined ultrastructurally in an effort to detect virus particles in the tumor cells. Newborn hamsters were inoculated sc with 0.2 ml viral material with a titer of 10^4 $TCID_{50}$/ml. Tumors appeared after 50-100 days; tumors 12-25 cm were studied, the larger ones being pseudocystous and the smaller ones, solid tumors. Histological investigation indicated that the tumor cells were similar to fibroblasts, which was confirmed by electron microscopy. Ultrastructurally, the tumor cells had comparatively deep invaginations and pockets with a central granulated structure surrounded by a double nuclear membrane. In some of the cells with nuclear invaginations there were myelinlike figures or irregular particles of different electron density surrounded by less electron-compact material. Nucleoli were frequently found attached to the nuclear membrane close to these invaginations and pockets, in which there were more ribosomes than in the remaining part of the cytoplasm; this is taken as a morphological expression of activation of the nuclear-cytoplasmic transport and metabolic processes. It is concluded that the particles found in the nuclei cannot be considered viral particles because of the absence of characteristic morphological features but they could be related to viral replication. It is suggested that BAV 3 transforms cells from the connective tissue, originating from the zone of application, into blastoma cells. (8 references)

3955 THE TRANSCRIPTIONAL ROLE OF HOST DNA-DEPENDENT RNA POLYMERASES IN ADENOVIRUS-INFECTED KB CELLS. (Eng.) Weinmann, R.; Raskas, H. J.; Roeder, R. G. *In: Tumor Viruses. Cold Spring Harbor Symposia on Quantitative Biology.* Cold Spring Harbor Lab. (Cold Spring Harbor, NY): vol 39, pp. 495-499; 1975.

Studies conducted on adenovirus-infected KB cells indicated that there were three types of RNA polymerases in these cells. RNA polymerase I activity was not detected late after infection, although the active form of the enzyme could still be isolated. It is suggested that viral infection interfered with the activity of this enzyme. RNA polymerase II synthesized the nuclear, high molecular wt, viral RNAs. RNA polymerase III transcribed the virus-associated 5.5S RNA and the 5S host RNA. Because their α-amanitin sensitivities for synthesis of specific viral RNA's coincided with the toxin sensitivities of the respective host RNA polymerases, it appeared that all three were host enzymes.

3956 CHROMATIN-LIKE ORGANIZATION OF THE ADENOVIRUS CHROMOSOME. (Eng.) Corden, J. (Dept. Biochemistry and Biophysics, Oregon State Univ., Corvallis, OR 97331); Engelking, H. M.; Pearson*, G. D. *Proc Natl Acad Sci USA* 73(2): 401-404; 1976.

The structure of the adenovirus core was characterized. Staphylococcal nuclease (nucleate, 3'-oligonucleotidohydrolase; EC 3.1.4.7) cleaved DNA within disrupted adenovirus particles into a regular series of fragments with a repeat unit of 200 base pairs. This pattern did not appear when DNA alone was digested, suggesting that the orderly arrangement of core polypeptides protects discrete regions of DNA from nuclease attack. The 23 x 10^6 dalton adenovirus DNA molecule can accommodate 180 units of roughly 200 base pairs. Based on the stoichiometry of core polypeptides, it was calculated that each repeat unit contains six copies of polypeptide VII and a single copy of polypeptide V. This model was based on proposals for the structure of eukaryotic chromatin. Very brief nuclease digestion generated 1800 base pair fragments (1/20 of the adenovirus chromosome). Although the adenovirus core contains no histones, it has a chromatin-like design.

3957 PHYSICAL MAPPING OF TEMPERATURE-SENSITIVE MUTATIONS OF ADENOVIRUSES. (Eng.) Grodzicker, T.; Williams, J.; Sharp, P.; Sambrook, J. *In: Tumor Viruses. Cold Spring Harbor Symposia on Quantitative Biology.* Cold Spring Harbor Lab. (Cold Spring Harbor, NY): vol. 39, pp. 439-446; 1975.

The genetic and physical maps of adenoviruses were aligned by locating the positions of temperature-sensitive (ts) mutants on the physical map. A major feature of this method is the isolation of recombinants from a cross between ts mutants of different adenovirus serotypes, rather than from crosses between mutants of the same serotype. Recombinant DNA was analyzed with restriction endonuclease. In all cases studied to date, the structures of the recombinant genomes derived by restriction enzyme analysis are consistent with each other and with the genetic map. This method of analyzing hybrid recombinants with restriction endonucleases has wide applications. It can be used to map mutations in any pair of viruses or DNA genomes that satisfy two requirements: that they can recombine and that their genomes differ enough to be distinguished by restriction endonuclease mapping. (23 references)

3958 HUMAN ADENOVIRUS INFECTION IN MONKEY CELLS: AN EXAMPLE OF HOST RESTRICTION AT A STEP LATE IN REPLICATION. (Eng.) Baum, S. G.; Fox, R. I. *In: Tumor Viruses. Cold Spring Harbor Symposia on Quantitative Biology.* Cold Spring Harbor Lab. (Cold Spring Harbor, NY): vol. 39, pp. 567-573; 1975.

The phenomenon of enhancement (increasing adenovirus replication in monkey cells by superinfection with SV40) was studied by examining each step in adenovirus replication as it is known to occur in permissive human cells and seeing if this process was carried out efficiently in the nonpermissive monkey cell. Several studies indicated that the block to efficient replication resided at some step following RNA synthesis but preceding translation of late viral proteins. It has been impossible to detect any difference between transcription occurring in enhanced and unenhanced infection, and no difference has been detected in the amount of polyadenylic acid sequences on unenhanced versus enhanced mRNA. However, a defect in the association of late RNA species with polyribosomes has been observed. Electrophoresis of polysome-associated RNA from adenovirus-infected KB cells and uninfected KB cells indicated that virus-associated RNA was polyribosome-associated in infected human cells and that there was no such material in the polysome fraction of uninfected cells. In the unenhanced infection, although virus-associated RNA was made, association of this late RNA species with polyribosomes was deficient. Thus, the behavior of virus-associated RNA appeared to be similar to that of other species of late RNA. Adenovirus was able to establish a chronic or latent infection in African green monkey kidney cells, and SV40 was able to make this latent state manifest at some time after the initial adenovirus infection. These results were not due to chronic low-yield shedding of infectious virus into the medium by a small number of cells. (30 references)

3959 REGULATION OF HERPESVIRUS GENE EXPRESSION IN BIOCHEMICALLY TRANSFORMED CELLS: INVESTIGATIONS OF VIRUS-SPECIFIED REGULATORY FUNCTIONS. (Eng.) Spear, P. G. (Dept. Microbiology, Univ. Chicago, 939 E. 57th St., Chicago, IL 60637); Leiden, J. M. *Bibl Haematol (Basel)* (43): 53-58; 1976.

Preliminary investigations of viral gene regulation were performed in a mouse L cell line that lacks a functional gene for thymidine kinase (tk) but carries the tk gene of herpes simplex virus type 1 (HSV-1). Synthesis of tk by LVtk+ cells was enhanced significantly by infection of LVtk+ cells with a mutant of HSV-1 lacking the tk gene (tk-HSV-^{1}). This enhancement varied with the multiplicity of infection, and no enhancement was observed at the highest multiplicity (10 PFU/cell). Antiserum against HSV-1 infected rabbit serum neutralized at least 75% of the tk activity in extracts of Ltk- cells infected with tk+HSV-1, whereas the tk activity from uninfected wild-type L cells could be reduced by only 29% by the antiserum. The same antiserum neutralized 70% of the tk activity in extracts from uninfected LVtk+ cells and 80% of the tk from infected LVtk+ cells that were exhibiting maximum enhancement of activity, indicating that the enzyme detected in the cells before infection was antigenically similar to or identical with the HSV-1 tk. Infection with tk-HSV-2 did not result in enhanced tk activity. L cell tk was not enhanced by tk-HSV-1 infection. Puromycin (50 μg/ml, added at 7 hr after infection) abolished enhanced tk activity, suggesting that the enhanced activity was the result of a newly synthesized enzyme and not increased activity of the existing enzyme. This conclusion was supported by the reversible inhibition of enhanced activity with cycloheximide, which inhibited activity; after its removal from the medium, enhanced activity comparable to the controls was observed. (12 references)

3960 METABOLISM OF GALACTOSE IN HERPES SIMPLEX VIRUS-INFECTED CELLS. (Eng.) Brennan, P. J. (Depts. Virology and Epidemiology, Baylor Coll. Medicine, Houston, TX 77025); Steiner, S. M.; Courtney, R. J.; Skelly, J. *Virology* 69(1): 216-228; 1976.

Some features of the metabolism of membrane-associated sugars in HSV-infected cells were investigated. Normal human embryo lung (HEL) and monkey kidney (BSC-1) cells grown in the presence of small amounts of [^{14}C]galactose incorporated most (about 70%) of the perchloric acid insoluble radioactivity into glucose; the remainder of the radioactivity was retained in galactose. Herpes simplex virus type 1 and 2 (HSV-1 and HSV-2)-infected cells rapidly lost the capacity to incorporate radioactivity into the insoluble [^{14}C]glucose, and they concomitantly developed an enhanced ability to incorporate the label into the galactose of glycoproteins. In addition, in infected cells, much of the radioactivity normally incorporated into soluble [^{14}C]glucose was instead channeled into glycolipid, i.e., glucosyl- and lactosylceramides. Pronase digestion and gel filtration indicated that the affected perchloric acid-insoluble glucose-containing material belonged to two classes; one was probably mucopolysaccharide or polysaccharide in nature, and the other contained a small glycopeptide. They were both associated with a 100,000 *g* particulate subcellular fraction. The basis of this aberration was investigated, and it is suggested that the phenomenon is at least partially due to the presence of virus-specific proteins in the infected cell which are not suitable substrates for the attachment of glucose units. Enhanced glycolysis or abnormal compartmentalization of nucleotide sugar pools in HSV-infected cells may also be involved. An examination of enveloped radioactive HSV-1 virions showed that only [^{14}C]galactose was present in the acid-insoluble fraction; [^{14}C]glucose was absent. The array of viral glycolipids was similar to that in infected cells with the presence of glucosylceramide, hematoside (GM$_3$) and some gangliosides (GM$_1$, Gp$_3$) among the recognized glycolipids.

3961 A PRELIMINARY REPORT ON INVESTIGATION OF ONCOGENIC POTENTIAL OF HERPES SIMPLEX VIRUS TYPE 2 IN CEBUS MONKEYS. (Eng.) Palmer, A. E. (Infectious Diseases Branch, Natl. Inst. Neurological Communicative Disorders and Stroke, Natl. Inst. Health, Bethesda, MD 20014); London, W. T.; Nahmais, A. J.; Naib, Z. M.; Tunca, J.; Fuccillo, D. A.; Ellenberg, J. H.; Sever, J. L. *Cancer Res* 36(2): 807-809; 1976.

The pathogenesis of HSV-2 in persistently reinfected female Cebus monkeys and its oncogenic potential in this genus was studied. A total of 301 female and 133 male Cebus monkeys were placed under study during a 3-yr period. Females were inoculated intradermally into the cervix every six mo; 225 received virus and 76 received control material. More than 50% of the animals were infected on primary inoculation, and a similar percentage was found on the first reinoculation. Males were housed with females at 1:1 to 1:3 ratios. Eighteen percent of the males exposed to virus-inoculated females became infected. To date, a total of 55 pregnancies have produced 14 live births and 8 abortions. The remaining 33 animals are still pregnant. No neonatal herpes simplex virus type 2 infections were identified. Cytological changes of mild (atypia) to moderate (dysplasia) anaplasia have persisted for 12 to 32 mo in 13 herpes simplex virus type 2-infected females. These animals received their first inoculation 14 to 50 months ago. Persistent anaplasia has not been found in control animals.

3962 HERPESVIRUS TYPE 2-RELATED ANTIGENS AND THEIR RELEVANCE TO HUMORAL AND CELL-MEDIATED IMMUNITY IN PATIENTS WITH CERVICAL CANCER.

(Eng.) Aurelian, L. (Depts. Lab. Animal Medicine and Microbiology, The Johns Hopkins University, Baltimore, MD 21205); Strnad, B. C. *Cancer Res* 36(2): 810-820; 1976.

The interaction of herpes simplex virus type 2 with its host was studied. If virus is to coexist with its host, a defined level of molecular interaction between host and viral gene products must exist. The association of AG-4 with active tumor growth and its identification as a minor virion protein, also exposed on the surface of the infected cell, was accomplished by investigating the association between AG-4 complement-fixing activity with VP-4, a virion protein with a molecular wt of 161,000 daltons. The modulation of the host immune response by the results of this interaction may play a significant role in cancer control. In these terms, the observation that antibody to AG-4 is a macroglobulin and that, therefore, immunity to AG-4 may be T-cell independent should be investigated.

3963 EXPERIMENTAL INFECTION OF SQUIRREL AND MARMOSET MONKEYS WITH ATTENUATED HERPESVIRUS SAIMIRI.

(Eng.) Falk, L. (Dept. Microbiology, Rush-Presbyterian-St. Luke's and Univ. Illinois Medical Centers, Chicago, IL 60612); Wright, J.; Deinhardt, F.; Wolfe, L.; Schaffer, P.; Benyesh-Melnick, M. *Cancer Res* 36(2): 707-710; 1976.

The progress of marmosets following inoculations with an attenuated strain of Herpesvirus saimiri (HVS) was monitored. HSV was propagated in vero cells for 3 passages at 39 C and cloned 3 times at 34 C. HVS was inoculated (1 x 10^6 plaque forming units; 0.1-0.5 ml) into cotton-topped marmoset and squirrel monkeys; all inoculated monkeys became infected as HVS was reisolated after their circulating lymphocytes were cultured with vero cells and measurable levels of antiviral antibodies developed (measured by immunofluorescence and/or neutralization tests). None of the inoculated monkeys developed any signs of overt disease and all inoculated monkeys have survived 9 to 14 months postinoculation. The attenuated virus appears to be genetically stable as virus isolated from an infected marmoset was passed 3 times in vitro and then inoculated into other marmosets, which became infected and remained clinically well. Marmosets latently infected with attenuated HVS were not protected when challenged with a large dose (770 plaque-forming units) of oncogenic HVS, although these marmosets survived about 3 times longer than did inoculated control marmosets.

3964 SOLUBLE COMPLEMENT FIXING ANTIGENS IN EPSTEIN-BARR VIRUS CELL SYSTEMS.

(Eng.) Twardzik, D. R. (NCI Frederick Cancer Res. Center, Bldg. 560, Frederick, MD 21701); Ablashi, D. V.; Easton, J. M.; Armstrong, G. R. *Bibl Haematol (Basel)* (43): 412-416; 1976.

The proteins of P3HR1 cell cultures that produce Epstein-Barr virus (EBV) were labeled by addition of ^{14}C-amino acids to the media. Supernatants of homogenates of these cells, when analyzed by glycerol density gradient centrifugation, showed a bimodal distribution in molecular weights of the newly synthesized protein corresponding to 35,000 and 150,000 daltons. Unfractionated ^{14}C-labeled P3HR1 protein was preincubated with EBV antiserum under conditions giving maximum precipitation, and a high molecular wt antigen-antibody complex formed that settled at the bottom of the tube. Proteins not reacting with the antiserum were not displaced. Complement-fixing (CF) activity was distributed under only one of the two peaks. Hydroxyapatite column chromatography of the labeled protein yielded six peaks, five of which contained over 95% trichloroacetic acid-insoluble material. Two of the peaks contained CF activity when they were tested with EBV antiserum. The molecular wt of these two antigens was 35,000 and 220,000 daltons. (15 references)

3965 COMPARATIVE STUDIES WITH DIFFERENT STRAINS OF EPSTEIN-BARR VIRUS IN OWL MONKEYS AND MARMOSETS.

(Eng.) Rabin, H. (Litton Bionetics, Inc., 5516 Nicholson Lane, Kensington, MD 20795); Pearson, G. R.; Wallen, W. C.; Neubauer, R. H.; Cicmanec, J. L.; Levy, B. *Bibl Haematol (Basel)* (43): 326-330; 1976.

The development of antibodies to viral capsid antigen (VCA), early antigen (EA), and Epstein-Barr virus-determined nuclear antigen (EBNA) by marmosets and owl monkeys after they have been inoculated with various strains of Epstein-Barr virus, and in vitro lymphocyte transformation studies with cells of various species are reported. Seroconversions to VCA, EA, and EBNA were 6/8, 0/8, and 0/8, respectively, when HR-1 was injected into *Saguinus fuscicollis* and *S. nigricollis*, and 2/2, 0/2, and 0/2, following injection of B95-8. Antibody conversions to VCA, EA, and EBNA in *S. oedipus* were 1/1, 1/1, and 0/1 after HR-1 and 2/2, 2/2, and 0/2 after B95-8. The corresponding conversions in *Callithrix jacchus* following B95-8 were 2/2, 2/2, and 0/2. In *Aotus trivirgatus* after HR-1, antibodies to VCA, EA, and EBNA were produced in 4/4, 0/4, and 0/4. In the same species following B95-8, the corresponding responders were 2/2, 1/2, and 1/2, and after At 531-H, the responders were 4/4, 2/4, and 1/4. One *C. jacchus* became debilitated 4 1/2 mo after B95-8 and was killed. At necropsy, a large white mass was present in the spleen with numerous smaller white nodules scattered throughout the liver and mesenteric lymph nodes. Histologic diagnosis was lymphoma. Lymphocyte transformation experiments indicated transformation of owl monkey lymphocytes with cells from 7/12 donor animals using B95-8, but no transformations with cells from eight donors using P3HR-1 virus. Neither

B85-8 virus (0/15 donors) nor HR-1 virus (0/5 donors) transformed lymphocytes from white-lipped marmosets. Lymphocytes from 10/14 gibbons were transformed by B95-8 virus, but with P3HR-1 virus, only 1/15 gibbons had cells that showed clumping and growth. (9 references)

3966 BIOCHEMICAL APPROACHES TO DETECTION OF EPSTEIN-BARR VIRUS IN HUMAN TUMORS. (Eng.)

zur Hausen, H. (Institut fuer Klinische Virologie der Universitaet Erlangen-Nuernberg, Loschgestrasse 7, 852 Erlangen, West Germany). *Cancer Res* 36(2): 678-680; 1976.

Biochemical studies were applied to the detection of Epstein-Barr virus (EBV)-DNA in human tumor cells. These studies resulted in the consistent demonstration of viral nucleic acid in African Burkitt's lymphoma biopsies and in epithelial tumor cells of nasopharyngeal carcinomas. The viral DNA resides within those cells regularly in multiple copies per cell. Besides these tumors, significant concentrations of EBV-DNA were detected in a German lymphoma patient revealing histological characteristics of Burkitt's lymphoma. Moreover, virus DNA was also found in a patient suffering from immunoblastic lymphadenopathy. More than 50 additional B-cell lymphomas and more than 40 biopsies from patients with Hodgkin's disease did not contain detectable amounts of EBV-DNA when tested by nucleic acid hybridization.

3967 TUMOR INCIDENCE IN VISNA VIRUS INOCULATED MICE. (Eng.)

Lycke, E. (Inst. Medical Microbiology, Sahlgrenska Sjukhuset, S-413 45 Goteborg, Sweden); Svennerholm, B. *Experientia* 32(4): 514-515; 1976.

The development of tumors in Swiss albino, BALB/c, CBA, and DBA mice inoculated with Visna virus was studied. The mice were injected ip or sc when newborn and examined 8-12 mo later. In the Swiss mice, 3 of 27 males and 30 of 39 females developed tumors, while 1 of 19 males and 5 of 25 females of the control Swiss mice developed tumors. The tumors were mainly mammary carcinomas (38 mice). None of the other strains showed an increased incidence of tumors from inoculation of the virus. The findings are consistent with preliminary experimental observations that Visna virus causes functional changes in humoral and cell-mediated immunity.

3968 FUCOLIPIDS IN NORMAL AND VIRUS-TRANSFORMED CELLS. (Eng.)

Steiner, S.; Gacto, M.; Steiner, M. R. *In: Cellular Membranes and Tumor Cell Biology.* Annual Symposium on Fundamental Cancer Research, 1975. (Baltimore: Williams and Wilkins Co.): pp. 309-324; 1975.

Studies of the fucolipids on the surface of a variety of virus-transformed cell lines and cultured human tumor cells are reviewed. Normal and virus-transformed cells were grown in medium supplemented with radioactive fucose, and autoradiograms of one-dimensional chromatograms were prepared. The virus-transformed cells showed a marked decrease in the chromatographically least mobile component (fucolipid IV), with a concomitant increase in the simpler ones. This decrease in fucolipid was seen in cell lines transformed by oncornaviruses, simian virus 40 (SV40), and herpes simplex viruses (HSV) and in temperature sensitive (ts) mutants of murine sarcoma virus (MSV) and SV40 at permissive temperatures. The reduced incorporation of radiolabeled fucose in fucolipid IV resulted from decreased synthesis, not from increased catabolism. The metabolic relationship among the fucolipids of normal and transformed rat cells was examined with the use of radioactive fucose in pulse-chase studies. The results suggest that fucolipid IV is a relatively stable end product of fucolipid metabolism in normal cells, and the more chromatographically mobile fucolipids are precursors. Studies to determine whether fucolipid synthesis varied as a function of cell density indicated that the synthesis of fucolipid IV is cell density-dependent in normal cells but in cells transformed by MSV (or murine leukemia virus), fucolipid synthesis is not influenced by cell density. Investigations with various SV40-transformed BALB/c cell lines and HSV-transformed hamster cells indicated that the degree of blocked fucolipid metabolism is positively correlated with the degree of oncogenicity of virus-transformed cells. (14 references)

3969 SURFACE PROPERTIES OF MAMMALIAN C TYPE VIRUSES. (Eng.)

Maruyama, K. (Chiba Cancer Center Res. Inst., Nitona-Cho, Chiba 280, Japan); Miller, M. F.; Hixson, D. C.; Wagner, S. H. *Bibl Haematol (Basel)* (43): 425-429; 1976.

Studies related to characterization of the surface components of mammalian C type viruses are reported. Indirect immunoferritin tests using antivirus sera indicated that virus particles in RFT and FL-74 cells (induced by Friend's leukemia virus and Theilen's feline leukemia virus, respectively) possess common surface antigens that are apparently absent on the surface of noninfectious, endogenous virus particles in NWR-1 cells. Some particles in RT-FeSV-II cells appeared to represent ''endogenous'' particles, because particles in NWR-1 cells were labeled by anti-RT-FeSV-II serum. Particles produced by RT-FeSV-II cells and exhibiting patchy ferritin labeling with anti-RFT and anti-FeLV-R sera may represent recombinants of inoculated FeSV-ST and ''endogenous'' virus genome present in rat cells. Results of indirect immunoferritin tests using antisera to viral proteins or glycoproteins indicated that the common surface antigens of Friend's leukemia virus and FeLV-T particles include the major envelope glycopeptides but not the major internal polypeptide. Results of F-lectin binding tests indicated that infection by leukemia or sarcoma virus results in changes of the lectin-binding pattern on the cell surface to a pattern resembling that on the viral envelope. The changes may result directly from viral genetic information or indirectly from alterations of cellular gene expression by virus infection. (7 references)

3970 ENDOGENOUS ECOTROPIC MOUSE TYPE C VIRUSES DEFICIENT IN REPLICATION AND PRODUCTION OF XC PLAQUES.

(Eng.) Rapp. U. R. (McArdle Lab. for Cancer Res., Univ. Wisconsin, Madison, WI 53076); Nowinski, R. C. *J Virol* 18(2): 411-417; 1976.

Endogenous ecotropic type C viruses were induced by iododeoxyuridine from nontransformed and chemically or spontaneously transformed clones of the C3H/10T1/2 cell line. Viruses produced by cells of certain transformed clones were N-tropic and formed large XC plaques. Infected cells did not form syncytia in contact with XC cells, even when they produced large amounts of DNA polymerase-containing particles. Although these XC^- viruses infected mouse cells with high efficiency, as shown by the induction of murine leukemia virus group-specific antigens in infected cells, virus production, as determined by DNA polymerase-containing particles, was extremely low. Upon growth in certain mouse cells, these replication-deficient, XC^- viruses converted to type C viruses that were similar, by XC assay, to N-tropic AKR virus (XC^+). The ability of endogenous type C viruses to replicate efficiently on the cells of origin may be an important determinant of their potential in their natural host.

3971 MECHANISM OF INDUCTION OF RNA TUMOR VIRUSES BY HALOGENATED PYRIMIDINES.

(Eng.) Besmer, P.; Smotkin, D.; Haseltine, W.; Fan, H.; Wilson, A. T.; Paskind, M.; Weinberg, R.; Baltimore, D. *In: Tumor Viruses. Cold Spring Harbor Symposia on Quantitative Biology.* Cold Spring Harbor Lab. (Cold Spring Harbor, NY): vol. 39, pp. 1103-1107; 1975.

The murine JLS V-9 cell line was used to study viral induction by halogenated pyrimidines. Exposure of JLS V-9 cells to iododeoxyuridine (IUdR) caused them to produce virus, but the induction was transient, with viral production decreasing slowly after removal of IUdR. IUdR induced no B-tropic murine leukemia virus (MuLV) production in Balb/c-3T3 cells, but did induce N-tropic MuLV production in NIH 3T3 cells and X-tropic virus production in NRK (rat) and SIRC (rabbit) cells. Synchronized JLS V-9 cells were pulsed with IUdR at various times during the cell cycle. Virus could only be induced in the middle part of the S phase, which means that the drug must be incorporated into a selected set of cellular genes. The toxic properties of bromodeoxyuridine (BUdR) for JLS V-9 cells could be overcome to a large extent by the inclusion of deoxycytidine (dC) in the medium. When JLS V-9 cells were continually maintained in the presence of BUdR, extensive virus production was evident for as long as 50 days, and even at 90 days virus was still being made. However, induction was not permanent, and the presence of BUdR was required. RNA was prepared from the nucleus and cytoplasm of induced and uninduced JLS V-9 cells, and the RNA preparations were tested for their content of virus-specific RNA by RNA-DNA hybridization. Induction caused about a tenfold increase in the amount of virus-specific RNA in the cytoplasmic fraction and a fivefold increase in the nuclear fraction. The uninduced cells also contained a significant fraction of virus-specific RNA in which almost all of the sequences of the DNA probe from the endogenous virus were represented. The data suggest that there is a critical segment of the genetic information of the cell which, when substituted with BUdR or IUdR, leads to a transcriptional derepression. (15 references)

3972 COMPARATIVE ASPECTS OF RNA TUMOR VIRUS ONCOGENESIS. COMPARATIVE STUDIES OF TYPE C, TYPE B AND M-PM ONCORNAVIRUS.

(Eng.) Dmochowski, L. (M. D. Anderson Hosp. and Tumor Inst., Univ. Texas System Cancer Center, Houston, TX 77030); Bowen, J. M.; Myers, B.; Priori, E. S.; Miller, M. F.; Seman, G.; Chan, J. C.; Dodson, M. L.; Scanlon, M.; Ohtsuki, Y.; Yoshida, H. *Bibl Haematol (Basel)* (43): 417-424; 1976.

The morphologic and antigenic properties of representatives of the three types of oncornavirus particles are summarized. Type C virus particles of mice are similar in size and appearance regardless of the strain of mice or origin of the tissue examined. However, extensive variation of the internal structure is apparent. The type C virus particles of the BALB/c/TEX mammary gland cell line behave like murine leukemia virus in their ability to induce giant cell formation in XC cells. An ultrastructural study of several type B particles in mammary tumors and in the milk of mice of high- and low-mammary cancer strains demonstrated an essential similarity in appearance and size and of their mode of development, regardless of origin. Internal structure, however, varied extensively. No antigenic differences were observed among viral particles from tumors or milk of the different high- and low-mammary cancer strains. No serologic cross-reactivity between types B and C viruses was observed, even when produced by the same cells. Ultrastructural studies of Mason-Pfizer monkey virus (M-PMV) revealed an essential similarity of structure whether the particles were grown in monkey embryo cells, in human cells, or in a simian mammary tumor. Considerable variation in internal structure was observed. HEp-2 and M-PMV particles closely resemble each other but differ markedly from similar forms of MuLV and MMTV particles. (28 references)

3973 VIRAL ONCOLOGY OF PROSTATIC CANCER.

(Eng.) Dmochowski, L. (Texas Medical Center, Houston, TX 77025); Horoszewicz, J. S. *Semin Oncol* 3(2): 141-150; 1976.

Evidence for the viral oncology of cancer of the prostate is reviewed. Ultrastructural studies of human prostatic neoplasia have demonstrated type C viral-like particles in 10/35 carcinoma cases and 1/8 benign hyperplastic specimens. Similar particles have been seen in other cancers such as comedocarcinoma of the breast. Varying numbers of type C viral particles were observed in the prostate glands of 22/34 mice of some mammary cancer strains, and the frequency of these particles increases with age. These particles could be a source of transmissible oncornaviruses. Immunologic studies indicate the presence, in the sera of patients with prostatic carcinoma, of antibodies to their own tumor cells. The nature of the antigens of prostatic carcinoma and the involvement of virus in their production have not been determined. Molecular hybridization studies have demonstrated the presence of viral information in the cellular DNA of prostatic carcinoma. Reverse transcriptase activity has also been found, and the viral information was transcribed to RNA. The etiologic relationship between viral components and human prostatic carcinoma is not known. However, sufficient circumstantial evidence exists to justify further investigation into a possible relationship. (47 references)

3974 IN VIVO INTERFERENCE OF VIRUS-INDUCED OSTEOSARCOMAS BY BENIGN BONE TUMOR VIRUS. (Eng.) Reilly, C. A., Jr. (Experimental Radiation Pathology and Oncology Group, Bldg. 340, Argonne Natl. Lab., Argonne, IL 60439); Finkel, M. P. *Bibl Haematol (Basel)* (43): 441-444; 1976.

Two bone tumor viruses isolated from a spontaneous osteosarcoma (FBJ virus) and osteoma (RFB virus) of CF1 mice provide a unique system for examining viral oncogenesis. Studies related to the interference of FBJ-induced osteosarcoma formation by RFB in newborn Swiss and CF1 mice are reported. When FBJ was given 24 hr prior to RFB, no interference was observed, but when the two viruses were administered simultaneously, there was some reduction in the number of osteosarcomas per mouse and some prolongation of life, but only an 8% reduction in the number of animals with tumors. There was significant interference in osteosarcoma formation when RFB was given 24 hr prior to FBJ (43% decrease in the number of animals with tumors, 43%-80% reduction in the number of tumors per mouse, and a twofold increase in mean time to death with tumor). When CF1 mice were foster-nursed by Swiss dams, the interference by RFB virus was even greater. Osteosarcoma incidences were lower in Swiss mice than in CF1 animals, both in terms of number of mice with tumors and number of tumors per mouse. With Swiss mice, the interference with osteosarcoma formation by RFB was the same whether sucklings were nursed by Swiss or CF1 dams. The enhancement of interference with tumor production in CF1 mice by nursing with Swiss dams suggests that an immunologic reaction is responsible for the interference, but the mechanism remains to be defined. (5 references)

3975 INHIBITION OF LYMPHOCYTE TRAPPING BY A PASSENGER VIRUS IN MURINE ASCITIC TUMORS: CHARACTERIZATION OF LACTIC DEHYDROGENASE VIRUS (LDV) AS THE INHIBITORY COMPONENT AND ANALYSIS OF THE MECHANISM OF INHIBITION. (Eng.) Mongini, P. K. A. (Stanford Univ. Sch. Medicine, Stanford, CA 94305); Rosenberg, L. T. *J Exp Med* 143(1): 100-113; 1976.

The effect of lactic dehydrogenase virus (LDV) on lymphocyte circulation was studied. Groups of female BALB/c mice were injected sc with SRBC, and 24 hr later 10^7 ^{51}Cr-labeled lymph node cells were injected iv. Antigen-draining and contralateral lymph nodes were excised after 24 hr, and the radioactivity in the nodes was determined. A significant increase in lymphocyte localization occurred in the peripheral lymph nodes draining the site of antigen injection (this was designated ''lymphocyte trapping''). A significant inhibition of trapping was observed in mice infused with cell-free ascitic fluid (3 5-ml doses) from 4 murine tumors (L4946, Ehrlich, ascitic lymphoma, and Krebs-2). Mice were then injected ip with 0.3 ml cell-free tumor ascitic fluid and tested for increases in lactic dehydrogenase 4 days later. The four murine tumors showed an approximately 8-fold increase in lactic dehydrogenase levels, indicating that each fluid that inhibited trapping contained LDV. Procedures used to eliminate infective LDV, such as dilution, passage of the tumor through irradiated Swiss Webster rats, ether fractionation, and UV irradiation, abolished the trapping inhibitory capacity of the fluids. Lymph nodes in mice with acute, but not chronic, LDV infections were inhibited from trapping. LDV did not appear to inhibit the capacity of circulating cells to be trapped, and as measured by mitogen responsiveness, the virus did not directly interfere with T-cell function. LDV may inhibit trapping by indirectly affecting the T cell, or by directly affecting the macrophage in which it replicates. The known characteristics of LDV infection may explain a number of reported immunosuppressive attributes of tumor-associated ascitic fluids.

3976 THERMAL TRANSITIONS IN VIRAL DNA (Meeting Abstract). (Eng.) Vizard, K. L. (Univ. Texas System Cancer Center, M. D. Anderson Hosp., Houston, TX 77025); Ansevin, A. T. *Biophys J* 16(21-part 2): 10a; 1976.

3977 MODIFICATIONS OF MORPHOLOGY AND FUNCTIONS OF THE GOLGI APPARATUS IN CELLS AT STAGES OF VIRUS-INDUCED TRANSFORMATION (Meeting Abstract). (Eng.) Leffingwell, T. P. (Cell Res. Inst., Univ. Texas, Austin, TX 78712); Whaley, W. G. *J Cell Biol* 70(2/Part 2): 85a; 1976.

3978 ISOLATION OF ROUS SARCOMA VIRUS TRANSFORMED CELLS HAVING LOW LEVELS OF PLASMINOGEN ACTIVATOR ACTIVITY (Meeting Abstract). (Eng.) Wolf, B. A. (Rockefeller Univ., New York, NY 10021). Goldberg, A. R. *J Cell Biol* 70(2/Part 2): 78a; 1976.

3979 DIFFERENTIAL EXPRESSION OF RELEVANT ROUS SARCOMA TUMOR ANTIGENS IN CULTURED CELLS (Meeting Abstract). (Eng.) Wainberg, M. A. (Lady Davis Inst. Medical Res., Jewish General Hosp., Montreal, Quebec, Canada); Israel, E.; Schwartz-Luft, E. *J Cell Biol* 70(2/Part 2): 42a; 1976.

3980 CHANGES IN SURFACE TOPOGRAPHY AND MICROFILAMENT ORGANIZATION UPON TRANSFORMATION OF CHICK EMBRYO FIBROBLASTS WITH ROUS SARCOMA VIRUS (Meeting Abstract). (Eng.) Wang, E. (Rockefeller Univ., New York, NY 10021); Goldberg, A. R. *J Cell Biol* 70(2/Part 2): 122a; 1976.

3981 RAMAN SPECTROSCOPY OF NORMAL AND ROUS-TRANSFORMED CELL MEMBRANES (Meeting Abstract). (Eng.) Carew, E. B. (Dept. Biology, Massachusetts Inst. Technology, Cambridge, MA 02139); Kaufman, H. W.; Robbins, P. W.; Stanley, H. E. *Biophys J* 16(21-part 2): 106a; 1976.

3982 MAREK'S DISEASE IN JAPANESE QUAIL--A PATHOLOGICAL, VIROLOGICAL AND SEROLOGICAL STUDY. (Eng.) Khare, M. L. (Dept. Animal Sciences, Thompson Hall, Cook Coll. Rutgers Univ., New Brunswick, NJ 08903); Grun, J.; Adams, E. V. *Poult Sci* 54(6): 2066-2080; 1975.

3983 EXPRESSION OF HEMOGLOBIN IN THE HYBRIDS FORMED BETWEEN MOUSE ERYTHROLEUKEMIA AND NUCLEATED OR ENUCLEATED MOUSE FIBROBLASTS (Meeting Abstract). (Eng.) Gopalakrishnan, T. V. (Lab. Biochemistry, NCI, NIH, Bethesda, MD 20014); Thompson, E. B. *J Cell Biol* 70(2/Part 2): 51a; 1976.

3984 IMMUNOLOGICAL STUDIES OF TUMORS INDUCED IN SHEEP BY INJECTING OVINE CELLS TRANSFORMED IN VITRO WITH FELINE SARCOMA VIRUS. (Eng.) Theilen, G. H. (Univ. California, Sch. Veterinary Medicine, Davis, CA 95616); Pedersen, N. C.; Hall, J. G. *Bibl Haematol* (43): 380-383; 1976.

3985 CELL RECOGNITION BY MALIGNANT MAMMARY EPITHELIUM (Meeting Abstract). (Eng.) Pitelka, D. R. (Dept. Zoology and Cancer Res. Lab., Univ. California, Berkeley, CA 94720). *J Cell Biol* 70(2/Part 2): 5a; 1976.

3986 HOST CELL FACTORS IN THE SURFACE OF THE MOUSE MAMMARY TUMOR VIRUS (Meeting Abstract). (Eng.) Sheffield, J. B. (Inst. Medical Res., Camden, NJ 08103); Lasfargues, E. Y.; Taraschi, N.; Daly, T. M. *J Cell Biol* 70(2/Part 2): 82a; 1976.

3987 ACTIN IN MAMMARY TUMOR VIRUS (Meeting Abstract). (Eng.) Damsky, C. H. (Wistar Inst., 36th & Spruce Sts., Philadelphia, PA); Tuszynski, G. P.; Sheffield, J. B.; Warren, L. *Biophys J* 16(21-part 2): 116a; 1976.

3988 MICROFILAMENT POLYMERIZATION AND VIRUS PRODUCTION (Meeting Abstract). (Eng.) Damsky, C. H. (Wistar Inst., Philadelphia, PA 19104); Sheffield, J. B.; Warren, L. *J Cell Biol* 70(2/Part 2): 117a; 1976.

3989 OBSERVATIONS ON THE INFECTIVITY OF THE MOUSE MAMMARY TUMOR VIRUS IN MINK LUNG CELLS (Meeting Abstract). (Eng.) Lasfargues, E. Y. (Inst. Medical Res., Camden, NJ 08103); Viadya, A. B.; Lasfargues, J. C.; Moore, D. H. *J Cell Biol* 70(2/Part 2): 81a; 1976.

3990 EFFECTS OF SODIUM BUTYRATE ON THE MORPHOLOGY, CYTOSKELETON, AND CELL SURFACE OF MURINE SARCOMA VIRUS-TRANSFORMED CELLS (Meeting Abstract). (Eng.) Altenburg, B. C. (Dept. Virology and Epidemiology, Baylor Coll. Medicine, Houston, TX 77030); Via, D. P.; Steiner, S. M. *J Cell Biol* 70(2/Part 2): 97a; 1976.

3991 EFFECT OF LOOSELY BOUND NONHISTONE CHROMOSOMAL PROTEINS AND RNA ON TRANSCRIPTION OF CHROMATIN FROM NORMAL AND SV-40-TRANSFORMED WI-38 CELLS (Meeting Abstract). (Eng.) Krause, M. O. (Dept. Biology, Univ. New Brunswick, Fredericton, N. B., E3B 5A3, Canada). *J Cell Biol* 70(2/Part 2): 7a; 1976.

3992 TEMPERATURE-DEPENDENT SURFACE CHANGES IN CELLS TRANSFORMED BY GROUP A MUTANTS OF SIMIAN VIRUS 40 (Meeting Abstract). (Eng.) Vollet, J. J. (Dept. Virology and Epidemiology, Baylor Coll. Medicine, Houston, TX 77030); Butel, J. S. *J Cell Biol* 70(2/Part 2): 51a; 1976.

3993 CELL CYCLE CHANGES INDUCED BY SIMIAN VIRUS 40 (Meeting Abstract). (Eng.) Lehman, J. M. (Dept. Pathology, Univ. Colorado Medical Center, Denver, CO 80220); Cram, L. S. *J Cell Biol* 70(2/Part 2): 97a; 1976.

3994 CONTRACTILE PROTEINS IN NORMAL AND TRANSFORMED FIBROBLASTS (Meeting Abstract). (Eng.) Schollmeyer, J. E. (Univ. Minnesota, Minneapolis, MN 55455); Wendelschafer-Crabb, G.; Furcht, L. T. *Biophys J* 16(21-part 2): 116a; 1976.

3995 CORRESPONDENCE BETWEEN T CELL SUPPRESSION AND ADLC TITERS DURING *HERPESVIRUS SAIMIRI* (HVS) INFECTION IN OWL MONKEYS. (Eng.) Wallen, W. C. (Litton Bionetics, Inc., 5516 Nicholson Lane, Kensington, MD 20795); Pearson, G. R.; Prevost, J.-M.; Neubauer, R. H.; Rabin, H. *Bibl Haematol* (43): 339-342; 1975.

3996 TYPE C PARTICLES IN NORMAL HUMAN PLACENTAS. (Ger.) Bierwolf, D. (Zentralinstitut fuer Krebsforschung, 1115 Berlin-Buch, Lindenberger Weg 70, East Germany); Rudolph, M.; Niezabitowski, A.; Jorde, A.; Bender, E.; Widmaier, R.; Graffi, A. *Arch Geschwulstforsch* 45(7): 628-633; 1975.

3997 PHENOTYPICALLY MIXED VESICULAR STOMATITIS VIRUS PARTICLES PRODUCED IN HUMAN TUMOR CELL LINES. (Eng.) Zavada, J.; Bubenik, J.; Widmaier, R.; Zavadova, Z. *In: Tumor Viruses. Cold Spring Harbor Symposia on Quantitative Biology.* Cold Spring Harbor Lab. (Cold Spring Harbor, NY): vol. 39, pp. 907-912; 1975.

3998 ELECTRON MICROSCOPIC INVESTIGATIONS OF A COMMON WART VIRUS AND A VIRUS ISOLATED FROM LARYNGEAL PAPILLOMAS. (Ger.) Ortel, S. (Medizinische Mikrobiologie und Epidemiologie, Martin-Luther-Universitaet Halle (Saale), Halle (Saale), East Germany). *Wiss Z Friedrich Schiller Univ Jena Math Naturwiss Reihe* 24(1): 9-14; 1975.

See also:

* (Rev): 3625, 3628, 3629, 3630, 3631, 3632, 3633, 3634, 3635, 3636, 3637, 3638, 3639, 3640, 3641, 3642, 3643, 3644, 3645, 3646, 3647, 3648, 3649, 3650, 3651, 3652, 3653, 3654, 3671, 3683, 3684, 3700, 3701, 3717, 3725, 3727
* (Chem): 3804
* (Phys): 3876
* (Immun): 4008, 4009, 4010, 4011, 4012, 4013, 4014, 4015, 4016, 4017, 4018, 4019, 4045
* (Path): 4060
* (Epid-Biom): 4115, 4118, 4119, 4121

3999 PURIFICATION AND TRANSLATION OF AN IMMUNOGLOBULIN λ CHAIN MESSENGER RNA FROM MOUSE MYELOMA. (Eng.) Honjo, T. (Faculty Medicine, Univ. Tokyo, Tokyo, Japan); Swan, D.; Nau, M.; Norman, B.; Packman, S.; Polsky, F.; Leder, P. *Biochemistry (Washington)* 15(13): 2775-2779; 1976.

The 500-fold purification of an mRNA encoding an immunoglobulin λ light chain derived from mouse myeloma tumor, RPC-20, is described. The purification involved the isolation of membrane-bound polysomes, oligo(dT)-cellulose chromatography, and sucrose gradient centrifugation under conditions favoring denaturation of polynucleotide complexes. The purified mRNA directed the cell-free synthesis of a polypeptide that was five or six amino acids longer than the mature form of the RPC-20 light chain. In addition, the mRNA migrated on electrophoresis as a band containing approximately 1,150 nucleotides, about 500 more than required to encode the mature form of the light chain. A portion of the region that therefore remains untranslated is assumed to be at both the 3' and 5' terminal ends. In this respect, the λ light chain mRNA is similar to both globin and other immunoglobulin mRNAs, in which extensive nucleotide sequences exist in the message that apparently do not contain structural information. These sequences are very important in the interpretation of hybridization studies using partial cDNA reverse transcripts. (37 references)

4000 IMMUNOGLOBULIN BIOSYNTHESIS BY THE MOPC 173 MOUSE MYELOMA TUMOR AND A VARIANT SPLEEN CLONE. (Eng.) Baumal, R. (Hosp. for Sick Children, Toronto, Ontario, Canada); Scharaff, M. D. *J Immunol* 116(1): 65-74; 1976.

IgG2a producing MOPC 173 tumor synthesized heavy (H) and light (L) chains and assembled them into H_2L_2 utilizing heavy chain dimers (H_2) and H_2L as the major precursors. Although the tumor cells secreted only H_2L_2, they synthesized excess L chains that appeared to be degraded and were not secreted. An L chain-producing variant arising spontaneously from the MOPC 173 tumor also failed to secrete any L chains. MOPC 173 tumor cells were cloned in the spleens of normal BALB/c mice and 1 of 23 spleen clones appeared to differ from the parent tumor in demonstrating a transient block in the assembly of H_2L_2 at the H_2 level, leading to delayed formation and secretion of H_2L_2 appreciable amounts of noncovalently bonded H_2L, an abnormal intracellular immunoglobulin species, H_4, and production of equimolar amounts of H and L chains. The patterns of immunoglobulin synthesis and assembly were also observed when tumor cells were studied in situ. Tryptic peptide mapping of the H and L chains produced by the MOPC 173 tumor and the variant spleen clone failed to demonstrate any differences in the H chains, but there were definite chemical differences in the L chains. Variant myeloma cells producing structurally altered immunoglobulins might continually be arising in myeloma tumors.

4001 MYELOMA WITH XANTHODERMA DUE TO AN IgG_λ MONOCLONAL ANTI-FLAVIN ANTIBODY. (Eng.) Farhangi, M. (Coll. Physicians and Surgeons, 630 W. 168th St., New York, NY 10032); Osserman*, E. F. *N Engl J Med* 294(4): 177-183; 1976.

An 80-yr-old woman with multiple myeloma developed xanthoderma, which was found to result from an IgG_λ (IgG Gar) monoclonal anti-flavin antibody. Purified IgG Gar was bright yellow, and the acid-dissociated chromophore was identified as riboflavin by chromatography and absorption spectroscopy. Native IgG Gar contained 1.45 M flavin/M IgG and increased to 2 M with addition of riboflavin to saturation. The flavin was localized to the Fab fragment and was bound to IgG Gar with high affinity. IgG Gar showed strongest affinities for riboflavin, flavin mononucleotide, and flavin adenine dinucleotide, and lower affinities for dinitrophenyl derivatives and naphthoquinone. The demonstration of hapten bound to the circulating monoclonal immunoglobulin in this case suggests the possibility of bound but colorless haptens on other myeloma proteins as well as on normal immunoglobulins.

4002 IgA AND IgM CYTOPLASMIC INCLUSIONS IN A SERIES OF CASES OF CHRONIC LYMPHOCYTIC LEUKAEMIA. (Eng.) Cawley, J. C. (Dept. Haematological Medicine, Hills Road, Cambridge CB2 2Q1, U.K.); Smith, J.; Goldstone, A. H.; Emmines, J.; Hamblin, J.; Hough, L. *Clin Exp Immunol* 23(1): 78-82; 1976.

Seventy-two cases of typical chronic lymphocytic leukemia were screened by electron microscopy for the presence of intracytoplasmic immunoglobulin crystals. Immunoglobulin inclusions were found in four cases. Immunofluorescent studies showed that the inclusions contained IgA in two cases and IgM in the other two patients. λ Light chain specificity was demonstrated in all four cases. The ultrastructure of the inclusions were identical in each patient except that in one of the IgA cases the inclusions were found in the perinuclear cistern in addition to the more usual location within cisternae of rough endoplasmic reticulum. Surface immunofluorescence showed μ heavy chains in the two cases displaying IgM crystal formation, but in the two IgA patients, no α heavy chains were demonstrable at the cell surface. The results show that inclusions are not restricted to IgM-synthesizing lymphocytes.

4003 PHYSICO-CHEMICAL CHARACTERIZATION OF MURINE J CHAIN. (Eng.) Barger, B. O. (Dept. Microbiology, 831 Bio Science Bldg., Univ. Georgia, Athens, GA 30602); Inman*, F. P. *Immunochemistry* 13(2): 165-171; 1976.

Some physical and chemical properties of mouse J chain were determined by experimentation. J chain was prepared from serum IgM of syngeneic BALB/c mice growing the HPC (AMF) 76 tumor. The molecular wt of the polypeptide was 20,000-24,000 daltons when determined by SDS-gel electrophoresis. When suspended in 5 M guanidine-HCl solution, however, its average molecular wt determined by low speed sedimentation equilibrium was 15.756 ± 642 daltons. The sedimentation constant (Sn_{20},w) of the J chain was 1.60S. Its partial specific volume was 0.707 ml/g. The amino acid composition of the mouse protein was very similar to that of human J chains and to that of some other species. Glucosamine was present but galactosamine was not detected.

4004 SURFACE IMMUNOGLOBULINS OF LYMPHOCYTES IN MOUSE PLASMACYTOMA: IV. EVIDENCE FOR THE PERSISTENCE OF THE EFFECT OF PLASMACYTOMA-RNA ON THE SURFACE IMMUNOGLOBULINS OF NORMAL LYMPHOCYTES IN VIVO AND IN VITRO. (Eng.) Bhoopalam, N. (Veterans Admin. West Side Hosp., P.O. Box 8195, Chicago, IL 60680); Yakulis, V.; Giacomoni, D.; Heller*, P. *Clin Exp Immunol* 23(1): 139-148; 1976.

Previous findings were confirmed that RNA extracted from murine plasmacytoma altered the character of the lymphocyte surface immunoglobulins (SIg) to express the idiotypic specificity of the Ig of the plasmacytoma from which the RNA was derived (cell conversion). RNA extracted from spleens of plasmacytoma-RNA-injected BALB/c mice also had converting activity, and (if injected into other mice) caused the appearance of RNA active in cell conversion of spleens of the second set of mice. This activity was lost only after two additional transfers. When splenic cells from animals 1 hr after injection with RNA extracted from MOPC 300, LPC-1 or MOPC 104E, were cultured for 7 days, the proportion of cells with the SIg specific for these tumors increased. The cell-converting activity of the RNA extracted from the cultured cells after 7 days incubation ('7-day' RNA) was higher than that of RNA extracted from cells after 1 hr incubation ('immediate' RNA). 'Seven-day' RNA could be used for sequential transfers without marked loss of activity in cell conversion for at least five transfers. The repetitive transferability of the phenomenon (by the injection of plasmacytoma-RNA) suggested the possibility of RNA replication in recipient cells.

4005 SURFACE IMMUNOGLOBULINS OF LYMPHOCYTES IN CHRONIC LYMPHOCYTIC LEUKEMIA. (Pol.) Leszko, B. (Instytutu Hematologii, ul. Chocimska 5, 00-957 Warsaw, Poland); Litwin, J.; Sylwestrowicz, T.; Pawelski, S. *Acta Haematol Pol* 7(1): 23-30; 1976.

The presence of IgM, IgG, and IgA on the surface of peripheral blood lymphocytes was studied with a modified fluorescence technique. Lymphocytes were obtained from 10 healthy subjects and 65 patients with chronic lymphocytic leukemia aged 44-84 yr, 30 untreated and 35 treated previously with cytostatic agents. The percentages for healthy subjects were 0-15 for IgG, 0-6 for IgM and 0-3 for IgA. The percentage of lymphocytes binding immunoglobulins on their surface was raised in almost all patients, compared to the healthy subjects. IgG predominated in 26/30 untreated patients and IgM, in 3/30; only 1 untreated patient showed the presence of no main Ig classes on the cell surface. Three showed primarily IgG (100%, 93%, 85%) as well as high levels of IgM (25%, 13%, 28%) and IgA (15%, 14%, 15%). Only IgG (from 30% to 100% of lymphocytes) was also present at high levels among treated patients. In one case, 90% of lymphocytes showed the presence of IgA, and two revealed the presence of no single main Ig class. Type of treatment, dosage, clinical care, and duration of the disease had no effect on the percentage of lymphocytes with various Ig classes bound to their surfaces in treated and untreated patients.

4006 CHARACTERIZATION OF L5178Y LEUKEMIC CELL WITH RAPID DEVELOP AND LOSE IMPLANTATION ABILITY. (Eng.) Sutkoff, D. R. (Univ. Rochester Sch. Medicine and Dentistry, Rochester, NY 14642); Bosmann, H. B. *Biochem Biophys Res Commun* 68(1): 277-283; 1976.

Two populations of L5178Y murine leukemic cells were studied for their implantation ability in BDF_1 mice. Implantation ability was measured by number of tumor nodules formed, liver wt, and day of death of the animals. Cells from a population grown from 10 yr in vitro had no implantation ability (no tumor nodules being formed when injected into the tail vein). After 30 days of growth in the peritoneal cavity of BDF_1 mice, these same cells were injected into the tail vein, and 10 days later had produced over 200 liver tumor nodules. When cells taken from these tumors were recultured for 60 days in vitro, they lost the acquired implantation ability, but regained it after another single peritoneal passage. L5178Y murine leukemic cells grown for 6 yr in ascites tumor cells were extremely tumorigenic: over 200 tumor nodules appeared in the liver after tail vein injection. These cells were not rendered less tumorigenic and did not lose their implantation ability by in vitro culturing for 60 days. Implantation ability could be a property of the cell's growth environment.

4007 ENHANCEMENT OF SKIN ALLOGRAFT SURVIVAL BY SOLUBLE ALLOANTIGEN IS SERUM-MEDIATED. (Eng.) Rao, V. S. (UCLA Sch. Medicine, Los Angeles, CA 90024); Bonavida, B. *Transplantation* 21(1): 42-50; 1976.

In previous experiments, H-2d mice treated before skin grafting with either soluble alloantigen prepared from H-2b cells or with serum obtained from similarly sensitized mice showed a significantly prolonged survival of H-2b skin allografts. This in vivo enhancement was paralleled by a depression of cell-mediated cytotoxicity to $[^{51}Cr]$H-2b target cells in vitro. Such in vitro and in vivo effects were abrogated in the present experiments when mice were repeatedly injected with soluble alloantigen before and after skin grafting, demonstrating that enhancing antibody present could be neutralized by this mode of antigen administration. The data suggest that serum factors present in mice pretreated with soluble alloantigen lead to specific blocking at the induction phase of the cell-mediated immune response and result in skin graft enhancement.

4008 VACCINATION OF NONHUMAN PRIMATES WITH KILLED ONCOGENIC HERPESVIRUSES. (Eng.) Laufs, R. (Hygiene Institut der Universitaet, D-34 Goettingen, Kreuzbergring 57, West Germany); Steinke, H. *Cancer Res* 36(2): 704-706; 1976.

The effect of killed HVS and HVA vaccines in protecting nonhuman primates against malignant lymphoma was investigated. Killed herpesvirus cancer vaccines were prepared by inactivation of oncogenic herpesviruses (Herpesvirus saimiri and Herpesvirus ateles) with heat and formaldehyde. The killed vaccines (not free of viral nucleic acid) were safe in 121 vaccinated monkeys of four different species. Some of these monkeys have been under observation for two years. The vaccines induced high titers of specific humoral antibodies in all vaccinated monkeys. The killed vaccines proved to be efficient against the im challenge with cell-free oncogenic herpesviruses. The challenged animals remained clinically well without signs of an infection and have now been under observation for one yr, while the nonvaccinated control monkeys died of malignant lymphoma 34 to 52 days after inoculation. The protection against H. saimiri could be passively transmitted with the serum of the vaccinated monkeys. The vaccination did not prevent but delayed tumor development after tumor cell transplantation. The resistance against the oropharyngeal route of infection remains to be determined.

4009 AN APPROACH TO C-TYPE VIRUS IMMUNOPREVENTION OF SPONTANEOUSLY OCCURRING TUMORS IN LABORATORY MICE. (Eng.) Kelloff, G. J. (Viral Carcinogenesis Branch, Natl. Cancer Inst., Bethesda, MD 20014); Peters, R. L.; Donahoe, R. M.; Ghazzouli, I.; Sass, B.; Nims, R. M.; Huebner, R. J. *Cancer Res* 36(2): 622-630; 1976.

The finding of C-type viruses or viral antigens in the spontaneous neoplasms of virtually all species where it has been sought, including the chicken, mouse, cat, hamster, viper, rat, pig, woolly monkey, gibbon ape, and possibly man, has suggested that these viruses are the specific etiological agents of these neoplasms. The demonstration of the oncogenicity of most of these virus isolates when inoculated into newborns of the same species provides further evidence of the role of the virus in the etiology of spontaneous tumors. Studies with two murine C-type viruses (Rauscher leukemia and Gross leukemia) a simian C-type virus, and an avian C-type virus (avian myeloblastosis virus) showed that the cell-mediated immune response of the animal includes type-specific, group-specific, and interspecies-specific reactivity. The mouse (BALB/c and NIH Swiss) gave a cell-mediated immune response to at least one of the polypeptides of the C-type virus, the gp69/71, whether this polypeptide was presented to the immune system of the mouse as whole virus, Tween-ether-treated virus, or a purified polypeptide. One measure of the effectiveness of the C-type virus vaccines was provided by immunization of the mouse with Rauscher leukemia virus preparation that induced resistance to challenge with both live Rauscher leukemia virus and a naturally occurring BALB/c leukemia virus. It is suggested that the C-type virus can act as an effective transportation antigen in syngeneic tumor cell lines resulting in the immunogenicity and loss of tumorigenicity of these cell lines.

4010 SURFACE MORPHOLOGY AND AGGLUTINABILITY WITH CONCANAVALIN A IN NORMAL AND TRANSFORMED MURINE FIBROBLASTS. (Eng.) Collard, J. G. (Dept. Experimental Cytology, The Netherlands Cancer Inst., Amsterdam 1004, The Netherlands); Temmink, J. H. M. *J Cell Biol* 68(1): 101-112; 1976.

Surface morphology of attached and suspended normal and transformed fibroblasts was studied using the scanning electron microscope. Normal murine fibroblasts (3T3) grew in vitro with widely extended leading lamellae. During most parts of the cell cycle, the surfaces of these cells were practically free of microvilli. When the cells rounded up for mitosis, their cell surfaces became adorned with many microvilli. In contrast, simian virus 40-transformed fibroblasts (SV3T3) grew more compact, and their cell surfaces remained smooth throughout the life cycle. When confluent 3T3 and SV3T3 cells were suspended with EDTA for agglutination assays, similar differences in surface morphology were found: 3T3 cells always had many microvilli; whereas most SV3T3 cells were essentially free of microvilli. The addition of concanavalin A did not influence the surface morphology of the suspended cells. The morphological differences could be important for the agglutination process of the normal and transformed 3T3 cells.

4011 INFLUENCE OF DNA-CONTAINING TYPE 16 ADENOVIRUS ON THE IMMUNE RESPONSE IN ADULT SYRIAN HAMSTERS. (Bul.) Ancheva, M. (MA, Oncological Center, Bulgaria); Gamburg, V. P. *Onkologia (Sofia)* 12(3): 118-123; 1975.

The effect of DNA-containing type 16 human adenovirus on the immune response was studied in adult Syrian male and female JCV hamsters, 1.5-2 mo old and never bred. The virus titer was 10^7 TCD_{50}/1 ml. It was administered through the heart (0.1 ml), ip (0.5 ml), and via the retroorbital sinus (0.1 ml). Noninfected human tissue was used for control. Immune responses were tested by the ability of the hamsters to produce antibodies against SRBC and paragrippal Sendai virus (live or dead). The animals were immunized ip with 5 x 10^8 or 10^9 SRBC/0.5 ml, or in the retroorbital sinus with 2 x 10^8/0.1 ml 5 or 9 days after the infection. The Sendai virus, 0.1 ml, was injected through the retroorbital sinus 9 days after infection. Hemagglutination titer (general and IgG) against SRBC was determined by the Takachi microtiter. The results show that the methods used to administer the adenovirus 16, SRBC immunization time, and antigen dosage had a negligible effect on the formation of antibodies. The total hemagglutinin and IgG hemagglutinin titers were the same in the experimental and control groups. The immunodepression observed in some experimental groups was mild and variable.

4012 IN VIVO AND IN VITRO MEASUREMENTS OF THE RELATIONSHIP OF HUMAN SQUAMOUS CARCINOMAS TO HERPES SIMPLEX VIRUS TUMOR-ASSOCIATED ANTIGENS. (Eng.) Hollinshead, A. C. (Lab. Virus and Cancer Res., Dept. Medicine, The George Washington Univ. Medical Center, Washington, DC 20037); Chretien, B. B.; Tarpley, J. L.; Kerney, S. E.; Silverman, N. A.; Alexander, J. C. *Cancer Res* 36(2): 821-828; 1976.

Unfiltered sera (224) were studied in a series of controlled, coded tests to determine the relationship of squamous carcinomas of the head and neck and cervix to the presence of complement-fixing antibodies to herpesvirus-tumor-associated antigens (HSV-TAA) in both tumor-bearing and cured patients. Sera from patients (90%) with squamous carcinomas had antibodies to HSV-TAA, in contrast to 11% of sera from patients with nonsquamous cancers and 4% of sera from normal individuals. The temporal relationship to Stage 1 laryngeal carcinomas suggests that HSV-TAA appearance precedes the immune defects. An in vitro correlate of the previously demonstrated specific delayed hypersensitivity reactions in controlled skin tests of squamous carcinoma patients with HSV-TAA were conducted. In leukocyte migration inhibition tests, the migration indices after incubation with HSV-TAA of peripheral blood leukocytes from patients with squamous carcinoma (x + 0.847) were in definite contrast to migration indices seen for normal leukocytes (x + 1.037) and patients with nonsquamous solid cancers (x + 1.03). Thus, these polypeptides elicit both humoral antibody response and cell-mediated reactivity.

4013 IMMUNE RESPONSE OF MONKEYS TO LYMPHOTROPIC HERPESVIRUS ANTIGENS. (Eng.) Pearson, G. R. (Viral Biology Branch, Natl. Cancer Inst., Natl. Inst. Health, Bethesda, MD 20014); Davis, S. *Cancer Res* 36(2): 688-691; 1976.

Herpesvirus saimiri induced leukemia and/or malignant lymphoma when inoculated into different species of nonhuman primates including marmosets and owl monkeys. No malignant disease has been recognized in the natural host for this virus, the squirrel monkey, although a high percentage of these monkeys are chronically infected with H. saimiri. Furthermore, one species of marmosets, as well as capuchin and some owl monkeys, did not develop lymphoma following experimental virus infection but developed a chronic infection similar to that noted in the natural host. The availability of susceptible and resistant species made it possible to attempt to delineate those humoral and cellular immune parameters that might correlate both with tumor induction and resistance. It is suggested that certain immune responses may be indicators of disease induction, and that others might play an active role in immunity to disease induced by the virus.

4014 AUTOGENOUS IMMUNITY TO ENDOGENOUS RNA TUMOR VIRUS: HUMORAL IMMUNE RESPONSE TO VIRUS ENVELOPE ANTIGENS. (Eng.) Hanna, M. G. (Basic Research Program, Natl. Cancer Inst., Frederick Cancer Res. Center, Frederick, MD 21701); Ihle, J. N.; Lee, J. C. *Cancer Res* 36(2): 608-614; 1976.

Autogenous immune sera from several strains of mice were tested for type-, group-, or interspecies-specific reactivities against leukemia virus envelope antigens and virus-induced cell surface proteins. The natural antibody of these test sera reacted with gp69/71, gp43, and p15 structural components on murine leukemia viruses including AKR, Friend, Rauscher, Moloney, and xenotropic BALB:virus-2. Comparable radioimmune titration curves were obtained in radioimmune precipitation assays. Competition experiments suggested that natural immune sera are predominantly type specific and only weakly cross-react with the Rauscher or Friend virus. Natural immune sera reacted with the virion envelope but not with the virus-induced cell surface antigen. An inconsistency appeared between the spectrum of virus-precipitating antibody and virus-neutralizing antibody. Although normal mouse serum readily neutralized xenotropic viruses (BALB:virus-2), only weak neutralization of the ecotropic viruses was achieved in vitro. It may be that autogenous immunity to murine leukemia virus is involved in the control of virus-mediated neoplasia, but more direct evidence is needed for this conclusion.

4015 ANTIBODY-DEPENDENT CELL-MEDIATED CYTOTOXICITY TO TARGET CELLS INFECTED WITH TYPE 1 AND TYPE 2 HERPES SIMPLEX VIRUS. (Eng.) Shore, S. L. (Center for Disease Control, Public Health Service, United States Dept. Health, Education, and Welfare, Atlanta, GA 30333); Black, C. M.; Melewicz, F. M.; Wood, P. A.; Nahmias, A. J. *J Immunol* 116(1): 194-201; 1976.

Antibody-dependent cell-mediated cytotoxicity (ADCC) was extended to include target cells acutely infected with herpes simplex type 1 virus (HSV-1) or herpes simplex type 2 virus (HSV02) in an in vitro system employing immune human serum and human blood mononuclear cells. The cytotoxic reaction was detectable after an hour of incubation and was complete between 4 and 8 hr. The amount of ADCC noted was directly proportional to the $\log_{10}$ of the effector:target cell ratio (E:T); ADCC was noted at E:T as low as 1:1. The mononuclear effector cell was present in the blood of both HSV immune and non-immune individuals. The immune serum factor was demonstrated to be an antibody with specificity for HSV membrane antigen(s) and was reactive with target cells infected with either of the 2 HSV types. The antibody rendered the mononuclear cell cytotoxic by sensitization fo the target cell (rather than by direct attachment to or ''arming'' of the mononuclear cell). The physiochemical properties of the antibody and its presence in cord blood demonstrated that it was an immunoglobulin of the IgG class.

4016 CELL-MEDIATED IMMUNITY TO FRIEND VIRUS-INDUCED LEUKEMIA: II. CHARACTERISTICS OF PRIMARY CELL-MEDIATED CYTOTOXIC RESPONSE. (Eng.) Ting, C.-C. (Natl. Cancer Inst., Bldg. 8, Room 219, Bethesda, MD 20014); Rodrigues, D.; Bushar, G. S.; Herberman, R. B. *J Immunol* 116(1): 236-243; 1976.

Primary cell-mediated cytotoxic response to a Friend virus-induced leukemia, FBL-3, in C57BL/6 mice was measured using the ^{125}I-iododeoxyuridine release assay. Ip inoculation of 1 x 10 FBL-3 cells produced progressive tumor growth (progressors); inoculation of as many as 5 x 10^6 FBL-3 cells produced only transient tumor growth (regressors); these mice would subsequently resist ip challenge of FBL-3 cells at 3 days after sc inoculation. The kinetics of the primary cell-mediated cytotoxic response of regressors was biphasic. Significant cytotoxicity could be detected at 3 to 5 days after sc inoculation of 5 x 10^6 FBL-3 cells, peaked at days 10 to 14, declined to a very low level or became undetectable around days 20 to 30; then the reactivity reappeared and persisted at least up to 60 days. In progressors, the kinetics of the cell-mediated cytotoxic response was similar to the regressors (but the reactivity was much lower). The cytotoxic response was found to be T cell dependent, during both the first peak (days 10 to 14) and the second peak (days 40 to 60). In adoptive transfer experiments, lymphocytes from regressors gave 90% protection against ip challenge of FBL-3.

4017 CELL-MEDIATED IMMUNITY TO FRIEND VIRUS-INDUCED LEUKEMIA: III. CHARACTERISTICS OF SECONDARY CELL-MEDIATED CYTOTOXIC RESPONSE. (Eng.)

Ting, C.-C. (Natl. Cancer Inst., Bldg. 8, Room 219, Bethesda, MD 20014); Kirchner, H.; Rodrigues, D.; Park, J. Y.; Herberman, R. B. *J Immunol* 116(1): 244-252; 1976.

By the use of the ^{125}I-iododeoxyuridine release cytotoxicity assay, the primary and secondary cell-mediated cytotoxic responses of C57BL/6 mice to FBL-3 cells, a syngeneic Friend virus-induced leukemia, were measured. The secondary cell-mediated cytotoxic response occurred more rapidly after challenge (within 3 days) than the primary response, and the levels of reactivity were considerably higher. The secondary cytotoxic reactivity of spleen cells was T cell dependent (being eliminated by pretreatment with anti-θ antibody plus complement). The secondary reactivity of peritoneal exudate (PE) cells was not entirely T-cell dependent. The specificity of the secondary cytotoxic response was analyzed by primary or secondary immunization with various tumor cells and by testing of cytotoxic lymphocytes against a variety of target cells. When spleen cells were used for testing, only tumor cells induced by Friend, Moloney, or Rauscher (FMR) leukemia viruses could produce secondary cell-mediated cytotoxic responses against FBL-3 cells. This correlated well with the specificity observed in the in vivo tumor transplantation protection studies. Spleen cells immune to FBL-3 had appreciable cytotoxicity against tumor cells induced by FMR viruses. The FBL-3 immune mice also gave significant protection against the challenge of FMR leukemias. When PE cells were used for testing, they gave a broader spectrum of activity.

4018 CELL-MEDIATED IMMUNITY TO LEUKEMIA VIRUS- AND TUMOR-ASSOCIATED ANTIGENS IN MICE.

(Eng.) Herberman, R. B. (Lab. Immunodiagnosis, Natl. Cancer Inst., Bethesda, MD 20014); Holden, H. T.; Ting, C.-C.; Lavrin, D. L.; Kirchner, H. *Cancer Res* 36(2): 615-621; 1976.

The role of cell-mediated immune reactions in resistance against the growth of leukemia cells in mice was investigated. Tumor models used (a Friend leukemia virus-induced transplantable tumor, FBL-3; a primary murine sarcoma virus (MSV)-induced tumor) were strongly antigenic; under some conditions, tumors regress completely. In mice (C57BL/6) with regressing FBL-3 tumors, cell-medited cytotoxicity was measured by release of [^{125}I]iododeoxyuridine. The response was biphasic, with an initial peak at 10 days and a 2nd peak after 30 days. A boost in reactivity could be elicited by later challenge with tumor cells. All reactivity was dependent on T-cells, being eliminated by treatment with anti-theta plus complement. The specificity of the reactions was consistent with Friend type-specific antigen plus broader, common antigens. In mice with regressing MSV tumors, strong cell-mediated cytotoxicity, measured mainly by release of ^{51}Cr was seen against RBL-5, a Rauscher virus-induced leukemia. A single peak of response occurred at about 14 days after virus inoculation. Upon later challenge with RBL-5 cells, a vigorous and rapid secondary response was elicited, mainly in the region of tumor challenge. This cytotoxic reactivity was dependent on T-cells. In addition, macrophage-mediated inhibition of leukemia cell growth in vitro was seen in this system at the time of peak tumor development. The ^{51}Cr release cytotoxicity was specific and directed primarily against an antigen, MEV-SA1, associated with mouse endogenous C-type viruses. The macrophage-induced growth inhibition appeared to be nonspecific. In both the FBL-3 and MSV tumor systems, protection against tumor growth could be adoptively transferred by immune lymphoid cells. Cell-mediated cytotoxic reactivity was found to occur naturally in most young mice. This activity was quite distinct from the experimentally elicited reactions, being mediated by N-cells (a subpopulation of lymphoid cells with no clearly identifiable cell surface markers). Natural cytotoxicity was also directed against antigenic specificities different from those recognized by the MSV-immune cells.

4019 PRESERVATION OF A T CELL-MEDIATED IMMUNE RESPONSE IN MAREK'S DISEASE VIRUS-INFECTED CHICKENS BY VACCINATION WITH A RELATED VIRUS. (Eng.)

Schierman, L. W. (New York Medical Coll., Valhalla, NY 10595); Theis, G. A.; McBride, R. A. *J Immunol* 116(5): 1497-1499; 1976.

The effects of vaccination and exposure to Marek's disease virus (MDV) on an in vivo T cell-mediated immune response and the humoral antibody response to SRBC were examined. In these studies, chickens from the inbred G-B1 strain and either attenuated MDV or a naturally occurring nonpathogenic herpesvirus from turkeys (HVT) were used. The results indicated that MDV was a potent immunosuppressive agent for a T cell-mediated immune response (an allograft reaction), possibly by necrobiosis of the thymus. There were no significant differences in the groups tested for IgG and IgM anti-SRBC. Vaccination with HVT interferes with the MDV-directed immunosuppression of thymus-derived cells, probably through preservation of the T cell response to MD tumor-associated antigens.

4020 SURFACE MARKERS AND PROGNOSTIC FACTORS IN ACUTE LYMPHOBLASTIC LEUKEMIA. (Eng.)

Tsukimoto, I. (Children's Hosp. Res. Foundation, Elland and Bethesda Ave., Cincinnati, OH 45229); Wong, K. Y.; Lampkin*, B. C. *N Engl J Med* 294(5): 245-248; 1976.

The surface markers on lymphoblasts from 37 patients with acute lymphoblastic leukemia were investigated. Spontaneous rosette formation with sheep erythrocytes (E rosettes) was used to identify T cells, and the presence of surface immunoglobulin was used to identify B cells. Eight patients had T-marker lymphoblasts; 28 had no markers (null lymphoblasts), and 1 patient had B-marker lymphoblasts. The 8 patients with T-marker acute lymphoblastic leukemia had massive leukemic infiltration, frequently a mediastinal mass, and a poor prognosis. The T-marker lymphoblasts had a weak or negative periodic acid-Schiff reaction and a very low antigenic stimulation to allogeneic lymphocytes. The association of T-marker lymphoblasts and some of the conventional poor-risk factors appears to be reliable in predicting a poor prognosis.

4021 LYMPHOCYTE SUBPOPULATIONS IN HODGKIN'S DISEASE.

(Eng.) Bukowski, R. M. (Dept. Hematology, Cleveland Clinic, 9500 Euclid Ave., Cleveland, OH 44106); Noguchi, S.; Hewlett, J. S.; Deodhar, S. *Am J Clin Pathol* 65(1): 31-39; 1976.

Lymphocyte subpopulations were studied in the peripheral blood, lymph nodes, and spleens in a group of 17 patients with untreated Hodgkin's disease. In 12 of 15 cases, the spontaneous rosette technique revealed diminished absolute levels of T-lymphocytes in the peripheral blood; however, this was correlated with total lymphopenia. No direct relationship between ''T-lymphopenia'' and diminished cellular immunity, as measured by phytohemagglutinin and pokeweed mitogen blast transformation, and delayed cutaneous hypersensitivy was demonstrated. In 8 lymph nodes involved histologically by Hodgkin's disease, a preponderance of T-lymphocytes was found when compared with a group of seven hyperplastic lymph nodes (78.2 ± 8.9% versus 54.5 ± 11.9%, $P < 0.01$). These latter data appear to be consistent with the hypothesis that the pathogenesis of Hodgkin's disease involves a cell-mediated immune response to a neoplastic (antigenic) element.

4022 THE USE OF B AND T MEMBRANE MARKERS IN THE CLASSIFICATION OF HUMAN LEUKEMIAS, WITH SPECIAL REFERENCE TO ACUTE LYMPHOBLASTIC LEUKEMIA.

(Eng.) Brouet, J.-C. (Hopital Saint-Louis, 75475 Paris Cedex 10, France); Preud'homme, J.-L.; Seligmann, M. *Blood Cells* 1(1): 81-90; 1975.

Some of the methods used to classify hematologic malignancies were evaluated according to their B or T cell origin. Cells from 90 leukemia patients were studied with use of surface immunoglobulins and IgG aggregates as B cell markers, spontaneous rosette formation with SRBC, and cytotoxic tests using heterologous antisera for identifying T cells. The vast majority of chronic lymphocytic leukemias (CLL) had a monoclonal B cell proliferation with a maturation block. However, biclonal proliferations were encountered, and a T cell origin was found in 5 out of 170 CLL. The abnormal cells in 14 patients with the Sezary syndrome were identified as T cells. In the majority of common acute lymphoblastic leukemias (ALL) no B or T markers were detected at the surface of the abnormal cells. These cells possessed no antigens reactive with the non-anti-B antibodies in antisera to CLL cells. in 30% of the cases the T nature of the blast cells appeared likely. In 11 other ALL patients a monoclonal B cell proliferation was found. These patients were usually not affected with common ALL and belonged mostly to two specific entities; in three cases the blastic proliferation supervent in patients previously affected with common CLL, and in six cases the blast cells possessed all the cytological features of Burkitt's tumor cells.

4023 CONCANAVALIN A POTENTIATES SYNGENEIC RESPONSE IN MURINE LYMPHOCYTES.

(Eng.) Ozato, K. (Dept. Embryology, Carnegie Institution Washington, Baltimore, MD 21210); Ebert, J. D. *J Exp Med* 143(1): 1-14; 1976.

In order to modulate the recognition processes occurring on lymphocyte membranes in mixed lymphocyte culture, responding cortisone resistant thymocytes or stimulating spleen cells (treated with mitomycin C) were pretreated with native concanavalin A (N-Con A) or succinyl-Con A (S-Con A). Significant cell proliferation was observed in syngeneic combinations when either the responding cells or the stimulating cells were so treated with Con A (although Con A pretreatment alone was never mitogenic). In allogeneic combinations, the proliferative response with Con A pretreatment of either partner on the third day was 5-7 times higher than in the normal mixed lymphocyte reactions. The triggering of proferation was dependent on the presence of spleen cells as the stimulating cells (thymocytes being much less effective) and the binding of Con A molecules to either one of the partners, the effect being abrogated by the specific inhibitor of Con A, α-mannopyranoside. The optimal concentration of S-Con A was about 2-fold that of N-Con A. Cultures in which either one of the partners was pretreated with Con A in allogeneic combinations showed a strong suppression (60-80% inhibition) in the subsequent generation of the cytotoxic lymphocytes (CL). The Con A concentration required to trigger a proliferative response corresponded to that for suppressing the generation of CL.

4024 THE IMMUNOSUPPRESSIVE MECHANISM OF AZATHIOPRINE: I. IN VITRO EFFECT ON LYMPHOCYTE FUNCTION IN THE BABOON.

(Eng.) Brown, T. E. (Naval Medical Res. Inst., Bethesda, MD 20014); Ahmed*, A.; Filo, R. S.; Knudsen, R. C.; Sell, K. W. *Transplantation* 21(1): 27-35; 1976.

The effect of azathioprine on in vitro baboon lymphocyte function tests was evaluated using the mitogen stimulation test, the mixed lymphocyte culture test, the migration inhibition factor test, the cell-mediated lymphocytotoxicity test, and the antibody dependent cell-mediated cytotoxicity test. Azathioprine inhibited phytohemagglutinin and Concanavalin A stimulation at lower concentrations than those required to inhibit pokeweed mitogen stimulation. It inhibited the mixed lymphocyte culture reaction with as little as 0.2 μg/culture in the microculture system. Azathioprine had no effect on the release of migration inhibition factor, the cell-mediated lymphocytotoxicity assay if presensitized cells were used, and the antibody-dependent cell-mediated cytotoxicity assay. However, azathioprine inhibited cell-mediated lymphocytotoxicity if it was added during in vitro sensitization and induction of killer cells. Azathioprine could inhibit those reactions requiring cellular division.

4025 LEUKOCYTE ADHERENCE INHIBITION. THE DETECTION OF CELLULAR RESPONSIVENESS AND SERUM 'BLOCKING' FACTORS IN MALIGNANCY. (Eng.) Bhatti, R. A. (Div. Urology, Cook County Hosp., Chicago, IL 60612); Ablin, R. J. *Allergol Immunopathol (Madr)* 3(5): 357-366; 1975.

The effectiveness of the leukocyte adherence inhibition (LAI) test in detecting cellular responsiveness and serum blocking factors was evaluated in two patients with prostatic carcinoma (case 1) and renal cell carcinoma (case 2). Three cultures were used that consisted of equal volumes of the patients' WBC plus: (a) normal human serum (NHS) of the same blood type (control); (b) tumor extract and NHS; and (c) tumor extract and the patient's serum. When the WBC were cultured with tumor extract and NHS, an interaction of antigen with presumably sensitized WBC occurred. There was no interaction between the patient's serum and tumor extract. In case 1, the presence of a cell-mediated response by the WBC to the tumor extract and the presence of a blocking factor in the serum were readily demonstrable. When the WBC and serum were reacted with an allogeneic extract, cross-reactivity was observed, indicating that the patient's WBC are sensitized to antigens apparently shared in common within tumors of the same type; however, there was an absence of blocking by the patient's serum. It was concluded that the LAI test is reliable. (20 references)

4026 CELLULAR RECEPTORS FOR LYMPHOTOXIN: CORRELATION OF BINDING AND CYTOTOXICITY IN SENSITIVE AND RESISTANT TARGET CELLS. (Eng.) Tsoukas, C. D. (Univ. California Sch. Medicine, San Francisco, CA 94143); Rosenau, W.; Baxter, J. D. *J Immunol* 116(1): 184-187; 1976.

The binding of ^{3}H-labeled lymphotoxin (LT) to both lymphotoxin-sensitive and -resistant cell clones was examined. Mouse L cells (clone 929) were used as a sensitive line from which 20 clones were obtained by soft agar cloning. Of these, one was markedly LT resistant; this line was recloned and used as a ''resistant'' line. Suspension cultures (35 ml each at 2×10^6 cells/ml) of both sensitive and resistant cells were incubated for 1 hr at 37 C with various amounts of nonradioactive LT. All cultures than received 600 cytotoxic units/ml ^{3}H-LT and were incubated for an additional hr at 37 C, after which radioactivity was determined. The sensitive clone had a low-capacity, high-affinity (''specific'') binding component, the curve of which closely followed the cytotoxicity curve of the lymphocyte mediator. The capacity of this binding component was calculated to be about 600 molecules of LT per cell. In addition, there was a low-affinity, high-capacity (''nonspecific'') binding component. In striking contrast, the high-affinity, low-capacity (''specific'') component was absent or greatly diminished from the resistant clone, whereas the low-affinity, high-capacity (''nonspecific'') component was present at a similar level as in the sensitive cells. These binding characteristics closely resemble those observed previously in studies with a variety of steroid hormones in steroid-sensitive and -resistant cell lines.

4027 IN VITRO GENERATION OF SUPPRESSOR CELL ACTIVITY: SUPPRESSION OF IN VITRO INDUCTION OF CELL-MEDIATED CYTOTOXICITY. (Eng.) Hodes, R. J. (Natl. Cancer Inst., Bethesda, MD 20014); Hathcock, K. S. *J Immunol* 116(1): 167-177; 1976.

When normal mouse spleen cells were cultured alone in vitro (precultured) for 3 to 7 days, these cells lost the ability to generate cell-mediated cytotoxicity (CML) during subsequent in vitro sensitization with allogeneic spleen cells, trinitrophenyl modified syngeneic spleen cells, or syngeneic tumor cells. These precultured cells, which were unable to generate CML, were also shown in mixing experiments to suppress the generation of CML by freshly explanted spleen cells. Suppression occurred at the sensitization phase of CML and not at the effector level; supernatants from suppressive precultured cells were not suppressive. Suppression was totally abrogated by the treatment of spleen cells with a T cell-specific rabbit anti-mouse brain serum and complement (RαMB + C) either before or after preculturing (suggesting that a T cell was essential both to the generation of suppressor activity and to its expression). Suppressor activity was entirely absent in precultured nylon wool column-nonadherent spleen cells, a T cell-enriched population containing most of the RαMB + C-sensitive cells in the spleen. Precultured nylon column-adherent cells (T cell-depleted) did have suppressive activity. A mixture of nylon-adherent and nylon-nonadherent cells was as suppressive after preculture as the precultured and unseparated spleen. The ability of nylon-adherent spleen cells to generate suppressive activity during preculturing was abrogated by treatment with RαMB + C. Therefore, the ''spontaneous'' generation of CML-suppressive activity was dependent upon a limited subpopulation of splenic T cells isolated in the nylon column-adherent fraction.

4028 EFFECTS OF CYCLAMATE CALCIUM ON THE IMMUNE RESPONSE IN RABBITS. (Eng.) Hampton, C. M. (Dept. Life Sciences, Southwest Missouri State Univ., Springfield, MO 65802); Myers*, R. L. *Arch Environ Health* 31(1): 51-53; 1976.

To determine the effect of cyclamate calcium on the humoral immune response in rabbits, 5 rabbits were given a 3% and 5 rabbits were fed a 5% water soluble ad libidum of cyclamate calcium. A third group served as controls. After 150 days, the animals were tested for their ability to respond immunologically to bovine serum albumin (BSA). Other aspects of immunity investigated were the presence or absence of neoplasia of the urinary bladder, spleen, thymus, and lymph nodes, the number of WBC and RBC, and cyclamate conversion and excretion in the urine. The results indicated that the 5% group had a delayed and suppressed response to BSA. No neoplasia was detected. This group had fewer RBC and excreted increased concentrations of cyclohexylamine in the urine. Because current research suggests that many tumors might arise as a result of a failure of the immune response to recognize neoplastic cells, it is concluded that cyclamate suppression of the antibody response may retard the tumor surveillance function of the immune system.

4029 THE IN VITRO SUPPRESSION OF ANTIGEN- OR MITOGEN-INDUCED DNA SYNTHESIS BY MURINE SPLEEN CELLS AFTER THE ADDITION OF FRESHLY PREPARED SYNGENEIC CELLS. (Eng.) Ficho, T. W. (Division Immunology, Hektoen Inst. Medical Res., 625 S. Wood St., Chicago, IL 60612); Baram*, P. *J Immunol* 116(2): 379-386; 1976.

The suppression of antigen- and mitogen-induced DNA synthesis by murine spleen cells in vitro was investigated. The addition of keyhole limpet hemocyanin (KLH), purified protein derivative (PPD), concanavalin A (Con A), phytohemagglutinin (PHA), or lipopolysaccharide (LPS) to 72-hr-old cultures of KLH-stimulated murine spleen cells resulted in the suppression of DNA synthesis assayed at 144 hr. When small numbers of freshly prepared KLH-PPD sensitive cells (SC) or non-sensitive cells (NSC) were added to these cultures at 72 hr the suppression of DNA synthesis was abrogated. However, the addition of larger numbers of KLH-PPD SC or NSC resulted in increased suppression of DNA synthesis. Large numbers of KLH-PPD SC or NSC could substitute for the second stimulant (KLH, PPD, Con A, PHA, or LPS) in suppressing DNA synthesis. The addition of fresh cells obtained from KLH-PPD-immunized mice were more effective in eliciting the subsequent suppression than were cells obtained from non-immunized mice. Cells obtained 30 to 75 days post immunization were most effective in suppressing DNA synthesis. It appeared that cultures receiving two signals exhibit marked suppression of DNA synthesis.

4030 MEMBRANE ASSOCIATED ANTIGENS OF HUMAN MALIGNANT MELANOMA II. LEUCOCYTE MIGRATION STUDIES WITH FORMALIN FIXED HUMAN MELANOMA CELLS. (Eng.) Suter, L. (Universitaets-Hautklinik, Von-Esmarch-Strass 56; D-44 Muenster, West Germany); Sorg, C.; Macher, E. *Z Immunitaetsforsch* 151(3): 242-249; 1976.

The usefulness of the leukocyte migration test from capillary tubes to assay cell-mediated immunity in melanoma patients was determined in formalin-fixed melanoma cells from established cell lines or from freshly excised tumors as an antigen source. From a group of 33 melanoma patients, 14 (42%) reacted positively when fixed cultured cells were used, whereas no positive reactions were found in 14 control donors. However, a considerable proportion of patients with chronic inflammatory processes of the skin reacted positively (4/17, 24%). A smaller proportion of positive reactions were found in melanoma patients when fixed melanoma cells from excised tumors were used (1/16, 6%). The positive results in the group with chronic inflammatory processes may be explained as reactions against melanoma-associated, but not melanoma-specific, antigens. Three possible reasons are given for the low frequency of positive reactions with cells from excised tumors: (a) the cells used display only a few of the antigenic determinants typical for malignant melanoma; (b) antigen expression is quantitatively lower; and (c) the surface antigens are covered by blocking factors.

4031 A COMMON CELL-TYPE SPECIFIC SURFACE ANTIGEN IN CULTURED HUMAN GLIAL CELLS AND FIBROBLASTS: LOSS IN MALIGNANT CELLS. (Eng.) Vaheri, A. (Dept. Virology, Univ. Helsinki, SF-00290 Helsinki 29, Finland); Ruoslahti, E.; Westermark, B.; Ponten, J. *J Exp Med* 143(1): 64-72; 1976.

Fibroblast surface antigen (SFA) is a high molecular wt glycoprotein first shown on the surface of cultured fibroblasts in fibrillar structures. SFA was shown to be present in cultured nonestablished astroglial cells. The glial and fibroblast SFAs were immunologically indistinguishable. Glial cells (three different nonestablished lines) contained more SFA per mg cellular protein than fibroblasts. SFA was located on cell surface in fibrillar striae that frequently extended out from the cell body. Fluorescence was also found intracellularly in the cytoplasm. Malignant gliomas (astrocytomas) established to grow to culture from human tumor material produced SFA into the growth medium but had very little (lines U-105 MG and U-343 MG) or no detectable (lines U-118 MG, U-251 MG, and U 343 MG-a) cell surface SFA. In cultures of the glioma cells, many cells (in particular those that appeared to be in the telophase stage) stained strongly positive for intracellular cytoplasmic SFA. Similar to fibroblasts transformed experimentally by oncogenic viruses, cells grown from naturally occurring human tumors (glioblastomas) produced SFA but lost the ability to retain it on cell surface.

4032 HL-A ANTIGENS IN PATIENTS WITH HODGKIN'S DISEASE. (Pol.) Maczynska-Rusiniak, B. (Instytutu Hematologii, Warsaw, Poland); Zupanska, B. *Acta Haematol Pol* 6(4): 267-271; 1975.

The frequency of HL-A antigens was established in 46 patients with Hodgkin's disease and was compared with the incidence in the Polish population at large. Statistical analysis was carried out by the chi square test. HL-A 1, 2, 3; W28 11, 9, 10; and HL-A5 7, 8, 12, 13, W5, W27, W10, W22, W15, and W17 antigens were determined. The most statistically significant difference between patients and healthy subjects was found with the HL-A5 antigen ($X^2 = 32.02$, $p = 0.005$), which appeared much more often in patients (43% vs 14.8%). The only other significant difference was found with W5, which was decreased in patients compared to healthy subjects (4.5% vs 13.5%; $X^2 = 7.08$, $p < 0.05$). The explanation for increased HL-A antigen in Hodgkin's disease may lie in the similar construction of this antigen and determinants of oncogenic viruses or in the connection between genes controlling transplant antigens and those controlling the system's immunologic response. (26 references)

4033 STIMULATION OF LYMPHOID CELLS FROM NORMAL AND IMMUNE MICE BY SYNGENEIC BALB/C PLASMA CELL TUMORS. (Eng.) Boyer, P. J. J. (Univ. California at Los Angeles Sch. Medicine, Univ. California, Los Angeles, CA 90024); Fahey, J. L. *J Immunol* 116 (1): 202-209; 1976.

The lymphoid cells from normal and immunized BALB/c mice could be stimulated in vitro by syngeneic plasma cell tumors (PCT) using a one-way mixed lymphocyte tumor interaction (MLTI) assay. The reactivity of normal spleen cells to syngeneic PCT contrasted with an absence of response to a number of other tumors. Maximal responses of normal cells to PCT were found to occur 5 days after the initiation of the cultures at an optimal responding:stimulating cell ratio of 1:2. MLTI activity of normal cells could not be blocked or enhanced by PCT myeloma protein products (indicating that MLTI reactivity was directed against non-idiotypic cell surface determinants). Lymphoid cells from immunized mice demonstrated increased MLTI responses to cells of the immunizing tumor but not to other PCT (indicating that the post-immunization MLTI responses were primarily to individual rahter than shared tumor cell surface antigens). Activity of both normal and immunized spleen cells was found to involve thymus-derived lymphocytes. However, the persistence of residual MLTI activity after treatment with anti-θ serum and complement implicated participation of non-θ antigen-bearing cells in MLTI reactivity. Lymphoid cells from unimmunized mice were capable of T cell-dependent reactivity to syngeneic PCT-associated antigens.

4034 IMMUNOLOGIC REACTIVITY OF B MICE RECONSTITUTED WITH VARIOUS NUMBERS OF SYNGENEIC BONE MARROW CELLS. (Eng.) Taylor, M. (Transplantation Biology Unit, Div. Surgical Sciences Clinical Res. Centre, Watford Road, Harrow, Middlesex HA1 3UJ, England); Simpson, E. *Transplantation* 21(1): 23-26; 1976.

A study was carried out to establish the useful experimental life of an immunologically deprived mouse and the lowest bone marrow reconstitution dose that gives maximal survival of the animals coupled with minimal T cell replacement. Adult thymectomized CBA mice were sublethally irradiated (950 rads) and reconstituted with various doses of syngeneic bone marrow. Immunological reactivity was assessed by allogeneic skin grafting at various intervals after reconstitution. It was found that reconstitution with high doses (1×10^7, 2×10^7 cells) of bone marrow was associated with an ability to reject allogeneic skin grafts and that this reactivity increased with time. Following rejection of a first skin graft, second grafts were rejected in an accelerated manner. These mice had measurable levels of Thy 1.2 positive cells in the spleen. In contrast, low-dose bone marrow reconstitution (1×10^6, 2×10^6 cells) produced mice that did not reject either first or second allogeneic skin grafts, and such mice had at most only very low levels of Thy 1.2 positive cells in their spleens. In the preparation of immunologically deprived mice for propagation of xenogeneic tumors, it is recommended either that low doses of syngeneic bone marrow be used for reconstitution or that the bone marrow be treated with anti-Thy 1.2 serum and complement before use.

4035 COMPARISON OF EARLY EMBRYONIC AND DIFFERENTIATING CELL SURFACES: INTERACTION OF LECTINS WITH PLASMA MEMBRANE COMPONENTS. (Eng.) Zalik, S. E. (Dept. Zoology, Univ. Alberta, Edmonton, Alberta, Canada T6G 2E1); Cook, G. M. W. *Biochim Biophys Acta* 419(1): 119-136; 1976.

Cells of the unincubated and those of primitive streak chick blastoderm, which were preparing for or were involved in morphogenetic movements, were agglutinated by wheat germ agglutinin, *Ricinus communis* agglutinin and concanavalin A, but not by fucose-binding protein. Agglutination of these cells with soybean agglutinin occurred only after neuraminidase treatment, while that induced by concanavalin A, wheat germ and *R. communis* agglutinins was not affected. Trypsin treatment of blastoderm cells had no effect on lectin-mediated agglutination. Cells derived from 10- and 12-day differentiating chick liver were agglutinated by wheat germ agglutinin only after trypsinization. Mechanically dissociated embryonic liver cells, which were not agglutinated, bound more ^{3}H-labeled wheat germ agglutinin per cell than trypsinized cells. This suggested that during differentiation, there might be a spatial reorganization of wheat germ agglutinin receptors within the plasma membrane. Membranes isolated from the above cell types were examined by analytical polyacrylamide gel isoelectric focusing and, in combination was affinity chromatography using wheat germ agglutinin conjugated to agarose, membrane material in the differentiating liver membrane, which bound to this lectin, was identified.

4036 INVOLVEMENT OF SIALIC ACIDS IN THE IMMUNOLOGICAL SPECIFICITY OF PLASMA MEMBRANE GLYCOPROTEINS. (Eng.) Prat, M. (Istituto di Anatomia, c.so Massimo d'Azeglio 52, 10126 Torino, Italy); Comiglio*, P. M. *Immunochemistry* 13(2): 97-102; 1976.

Solubilized membrane antigens (SMA) from a mouse plasma cell tumor wre used as immunogens to prepare rabbit antisera. Cells from a mineral-oil induced plasmacytoma (MOPC-460) were maintained in syngeneic Balb/C mice by serial sc transplantations. Antisera were tested against native SMA or SMA desialylated with *Vibrio cholerae* neuraminidase. Radioimmunoprecipitation showed that removal of sialic acids lowered the precipitation curves. When direct measurement of antigen antibody reaction was performed by a solid-state competitive binding radioimmunoassay, desialylated SMA were only partially able to inhibit the binding of radiolabeled antibodies to native antigens. Indirect immunofluorescence experiments showed that adsorption of the antisera with neuraminidase-treated living plasmacytoma cells did not completely remove antibodies directed against the cell surface.

It is suggested that sialic acids are directly or indirectly involved in the formation of a small but definite number of antigenic determinants of membrane glycoproteins.

4037 RADIOIMMUNOASSAY OF HUMAN C-REACTIVE PROTEIN AND LEVELS IN NORMAL SERA. (Eng.) Claus, D. R. (Rush-Presbyterian-St. Luke's Medical Center, Chicago, IL 60612); Osmand, A. P.; Gewurz, H. *J Lab Clin Med* 87(1): 120-128; 1976.

C-reactive protein (CRP) has been considered an acute-phase protein appearing only during reactions of tissue injury or inflammation. Reported was the quantitation of CRP levels in normal adults and neonates made possibly by the development of a sensitive and precise radioimmunoassay for human CRP that enabled the detection of 3 mg/ml. Serum levels in 153 healthy blood donors ranged from 68 to 8,200 ng/ml with a median value of 580, (mean = 1,340). CRP levels in 24 normal cord serum samples ranged from 10 to 370, with a median value of 70 (mean = 109). CRP levels in 246 individuals evaluated for autoimmune diseases ranged to 256,000, with a median value of 13,000. No individual lacking CRP was detected. Therefore, CRP was considered a component of normal serum that only increased dramatically in concentration during inflammation.

4038 IMMUNOBIOLOGY OF OPERABLE BREAST CANCER: AN ASSESSMENT OF BIOLOGIC RISK BY IMMUNOPARAMETERS. (Eng.) Wanebo, H. J. (Depts. Surgery, Pathology, Medicine, Biostatistics Lab., Memorial Sloan-Kettering Cancer Center, 1275 York Ave., New York, NY 10021); Rosen, P. P.; Thaler, T.; Urban, J. A.; Oettgen, H. F. *Ann Surg* 184(3): 258-267; 1976.

4039 BROWN ADIPOSE TISSUE: RELATIONSHIP TO CELL-MEDIATED IMMUNITY. (Eng.) Jankovic, B. D. *In: Host Defense Against Cancer and Its Potentiation. Proceedings of the 5th International Symposium of The Princess Takamatsu Cancer Research Fund, Tokyo, 1975.* (Baltimore: Univ. Park Press): pp. 303-316; 1975.

4040 TUMOR CELL-HOST CELL INTERACTIONS IN LYMPH NODES OF PATIENTS WITH MALIGNANT MELANOMA (Meeting Abstract). (Eng.) Rowden, G. (McGill Univ. Cancer Res. Unit, Montreal, P. Q., Canada); Lewis, M. G.; Jerry, L. M. *J Cell Biol* 70(2/Part 2): 3a; 1976.

4041 IMMUNOLOGIC CROSS-REACTIVITY OF ANTIBODIES TO A SYNTHETIC UNDECAPEPTIDE OF CARCINOEMBRYONIC ANTIGEN WITH THE INTACT PROTEIN AND WITH HUMAN SERA (Meeting Abstract). (Eng.) Arnon, R. (Dept. Chemical Immunology, Weizmann Inst. Science, Rehovot, Israel); Bustin, M.; Calef, E.; Chaichik, S.; Haimovich, J.; Novik, N.; Sela, M. *Isr J Med Sci* 11(12): 1399; 1975.

4042 NORMAL IMMUNOSUPPRESSIVE PROTEIN AND IMMUNOLOGIC SURVEILLANCE (Meeting Abstract). (Eng.) Ovadia, H. (Dept. Immunology, Hebrew Univ.-Hadassah Medical Sch., Jerusalem, Israel); Hanna, N.; Nelken, D. *Isr J Med Sci* 11(12): 1398; 1975.

4043 IMMUNODEFICIENCY AND EPILEPSY (Letter to Editor). (Eng.) Kanoh, T. (Dept. Internal Medicine, Kyoto Univ., Kyoto 606, Japan); Uchino, H. *Lancet* 1(7964): 860-861; 1976.

4044 INFLUENCE OF ALLOGENEIC THYMOCYTES ON RADIOLEUKEMOGENESIS IN ARK-T1ALD MICE. (Fre.) Legrand, E. (INSERM, Unite 117, Fondation Bergonie, 180, rue Saint-Genes, 33076 Bordeux, France); Sankar-Mistry, P.; Kressmann, M. C. *Biomedicine* 23(4): 303-310; 1975.

4045 EVIDENCE FOR THE COATING OF SPECIFIC ANTIGENIC SITES ON POLYOMA-INDUCED TUMOR CELLS DURING GROWTH IN A SYNGENEIC HOST (Meeting Abstract). (Eng.) Ran, M. (Dept. Microbiology, George S. Wise Center for Life Sciences, Tel Aviv Univ., Tel Aviv, Israel); Witz, I. P. *Isr J Med Sci* 11(12): 1397-1398; 1975.

4046 MEMBRANE-RELATED ANTIGENS IN PLEURAL FLUIDS OF PATIENTS WITH BREAST CANCER (Meeting Abstract). (Eng.) Gorsky, Y. (Lautenberg Center General Tumor Immunology, Hebrew Univ.-Hadassah Medical Sch., Jerusalem, Israel); Sulitzeanu, D. *Isr J Med Sci* 11(12): 1399-1400; 1975.

4047 DEGRADATION OF ANTITUMOR ANTIBODIES BY LYSOSOMAL ENZYMES: THE BLOCKING EFFECT OF THE DEGRADATION PRODUCTS (Meeting Abstract). (Eng.) Keisari, Y. (Dept. Microbiology, George S. Wise Center for Life Sciences, Tel Aviv Univ., Tel Aviv, Israel); Witz, I. P. *Isr J Med Sci* 11(12): 1398-1399; 1975.

4048 EVIDENCE FOR BINDING OF NONRELATED ANTIBODY BY TA$_3$ TUMOR CELL POPULATIONS (Meeting Abstract). (Eng.) Braslawsky, G. (Dept. Microbiology, George S. Wise Center for Life Sciences, Tel Aviv Univ., Tel Aviv, Israel); Yaakubowicz, M.; Frensdorff, A.; Witz, I. *Isr J Med Sci* 11(12): 1398; 1975.

4049 AUTO-ANTIBODIES IN THE SERA OF PATIENTS WITH NASOPHARYNGEAL CARCINOMA. (Eng.) Yoshida, T. O.; Yasuda-Yasaki, Y.; Utsumi, K. R. *In: Oncogenesis and Herpesviruses II. Part 2: Epidemology, Host Response and Control. Proceedings of a Symposium held in Nuremberg, Federal Republic of Germany, 14-16 October 1974.* International Agency for Res. on Cancer. (Lyon, France): pp. 259-273; 1975.

4050 TISSUE SPECIFIC ANTIBODIES AGAINST HUMAN TISSUE DEHISTONIZED CHROMATINS (Meeting Abstract). (Eng.) Chiu, J. F. (Dept. Biochemistry and Pathology, Vanderbilt Univ. Sch. Medicine, Nashville, TN 37232); Chytil, F.; Rogers, L. W.; Hnilica, L. S. *J Cell Biol* 70(2/Part 2): 124a; 1976.

4051 EXPRESSION OF MEMBRANE ABH ANTIGENS ON TRANSFORMED HUMAN CELLS (Meeting Abstract). (Eng.) Kuhns, W. J. (Dept. Pathology, New York Univ. Sch. Medicine, New York, NY 10016); Pann, C.; Kanra, G. *J Cell Biol* 70(2/Part 2): 11a; 1976.

4052 ANTI-IMMUNOGLOBULINS IN MULTIPLE MYELOMA AND BENIGN MONOCLONAL HYPERGLOBULINEMIA. (Ger.) Schedel, I. (Medizinische Hochschule Hannover, Hannover, West Germany); Fink, P. C.; Kalden, J. *Blut* 32(4): 275-278; 1976.

4053 TRANSFORMED LYMPHOCYTES. LECTIN-INDUCED MORPHOLOGICAL CHANGES (Meeting Abstract). (Eng.) Temminck, J. H. (Dept. Electron Microscopy, Netherlands Cancer Inst., Amsterdam, Netherlands). *J Microsc Biol Cell* 24(2/3): 196-197; 1975.

4054 ANALYSIS OF CELL POPULATIONS IN MURINE LYMPHATIC LEUKEMIAS (Meeting Abstract). (Eng.) Linker-Israeli, M. (Dept. Chemical Immunology, Weizmann Inst. Science, Rehovot, Israel); Haran-Ghera, N. *Isr J Med Sci* 11(12): 1380; 1975.

4055 RELATIONSHIP BETWEEN T ROSETTE-FORMING CELLS AND CELLULAR IMMUNITY IN VARIOUS HUMAN DISEASES (Meeting Abstract). (Eng.) Shohat, B. (Clinical Lab., Beilinson Medical Center, Petah Tikva, Israel); Joshua, H. *Isr J Med Sci* 11(12): 1391; 1975.

4056 IONOPHORE A-23187 INDUCED HISTAMINE RELEASE FROM RAT MAST CELLS AND RAT BASOPHIL LEUKEMIA (RBL-1) CELLS. (Eng.) Siraganian, R. P. (Building 10, Room 2B-12, NIH, Bethesda, MD 20014); Kulczycki, A., Jr.; Mendoza, G.; Metzger, H. *J Immunol* 115(6): 1599-1602; 1975.

4057 MODULATION OF AGGLUTINABILITY BY ALTERATION OF THE SURFACE TOPOGRAPHY IN MOUSE ASCITES TUMOR CELLS (Meeting Abstract). (Eng.) Oppenheimer, S. B. (Dept. Biology, California State Univ., Northridge, CA 91324); Bales, B. L.; Brenneman, G.; Knapp, L.; Lesin, E. S.; Neri, A.; Pollock, E. G. *J Cell Biol* 70(2/Part 2): 6a; 1976.

4058 T AND B BLOCKING FACTORS IN HODGKIN'S DISEASE (Meeting Abstract). (Eng.) Bentwich, Z. (Dept. Medicine A, Hebrew Univ.-Hadassah Medical Sch., Jerusalem, Israel); Cohen, R.; Brautbar, C. *Isr J Med Sci* 11(12): 1382; 1975.

See also:

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* (Chem): 3744, 3780, 3791, 3814
* (Phys): 3831, 3853
* (Viral): 3896, 3902, 3907, 3910, 3917, 3918, 3923, 3930, 3939, 3962, 3963, 3964, 3965, 3975, 3982, 3984
* (Path): 4080
* (Epid-Biom): 4111, 4144

4059 ERYTHROCYTE ENZYME DEFECTS IN PANCYTOPENIA AS PROGNOSTIC FACTORS FOR ACUTE LEUKEMIA. (Eng.) Nowicki, L. (Zentrum der Inneren Medizin der J. W. Goethe-Universitaet, Abteilung fuer Hamatologie, 6 Frankfurt/Main, West Germany). *Adv Biosci* 14: 563-565; 1975.

Defects in erythrocyte enzyme activity were investigated for their possible prognostic relationship to the development of leukemia. In a study of 7 patients with Di Guglielmo's syndrome and 10 patients with acute leukemia, significant decreases in erythrocyte pyruvate kinase and/or glutathione reductase activities were found in 4 patients from each group. Among 22 patients with paroxysmal nocturnal hemoglobinuria, 11 showed decreased activities of one or both of the above erythrocyte enzymes. Among 63 patients with pancytopenia, 10 (2 males, 8 females, average ages 51.0 and 44.9 yr, respectively) showed glutathione reductase deficiency (55.6% of normal); 21 patients (9 males and 12 females, average ages 44.4 and 54.9 yr, respectively) exhibited pyruvate kinase deficiency (44.3% of normal); and 12 patients (6 males and 6 females, average ages 42.4 and 56.5 yr, respectively) were deficient in both glutathion reductase (55% of normal) and pyruvate kinase (44.4% of normal). Nine of the patients with enzyme defects died within 1 yr after diagnosis, whereas only 1 of the 20 without erythrocyte enzyme disorders died. Four patients out of the 43 with enzyme defects developed acute leukemia, but only 1 of the 20 without enzyme defects developed the same condition. Pancytopenia appears to resemble a possible prestage of leukemia, and the incidence of leukemia rises twofold if pancytopenia is linked with erythrocyte enzyme disorders. Thus, decreased activities of either or both of the above enzymes appear to be useful as prognostic factors for leukemia. (no references)

4060 VIRUS-LIKE PARTICLES IN CHRONIC LYMPHATIC LEUKEMIA: ULTRASTRUCTURAL FINDINGS. (Eng.) Spremolla, G. (Istituto di Clinica Medica Generale e Terapia Medica I dell'Universita di Pisa, Italy); Cioni, C. *Haematologiga* 59(4): 467-479; 1974.

The ultrastructural aspects of the bone marrow cells from a 50-yr-old male patient with chronic lymphocytic leukemia (CLL) were investigated. The disease had an unusual onset characterized by moderate leukocytosis with a predominance of granulocytes and some metamyelocytes and myelocytes. The marrow aspirate showed normal cells, a moderate number of lymphocytes, and an abundance of myeloid elements and promyelocytes, suggesting an initial stage of myeloblastic leukemia. One month later, the hematological picture had changed from a suspected myeloid to lymphatic leukemia. The cell population was comprised mostly of lymphoid cells that had an irregular outline showing several small indentations of the plasma membrane. The nucleus was irregular with deep invaginations. In the cytoplasm there were larger than normal mitochondria, a distinct Golgi apparatus, and cisterna of endoplasmic reticulum. In the cytoplasm of some cells, virus-like particles previously observed in cases of chronic and acute leukemia were found. The particles, found both isolated and in groups forming racemose structures, presented an electron-dense nucleoid limited by a single or double membrane. Their peculiar feature was a fairly electron-dense filament in the perinuclear cisterna apparently originating from the nuclear material itself, which could not be measured. On occasion, these filaments were found in cytoplasm and the cisternae of the endoplasmic reticulum near a group of virus-like particles. (61 references)

4061 A THYMUS-SPECIFIC ENZYME IN ACUTE LYMPHOBLASTIC LEUKEMIA CELLS. (Eng.) McCaffrey, R. (Dept. Medicine, Children's Hosp. Medical Center, Boston, MA 02115); Smoler, D. F.; Harrison, T. A.; Baltimore, D. *Adv Biosci* 14: 527-534; 1975.

A unique DNA synthetic enzyme, terminal deoxynucleotidyl transferase, was found in the circulating leukemic cells from five of seven patients (ages 5-14 yr) with childhood acute lymphoblastic leukemia. Clinically, there was little that distinguished the children with the enzyme from those without it. All seven showed high WBC counts (30,000-700,000 mm^3). Four of the five patients with cells showing the activity have achieved remission on therapy with vincristine and prednisone induction. Terminal deoxynucleotidyl transferase activity has previously been considered a special property of thymic tissue, and its presence in the cells of the five children suggests that the leukemic cells may be thymic lymphocytes blocked at an early level of differentiation in the thymus. The immediate source of the leukemic cells seeding the peripheral blood and bone marrow would, in this scheme, be the thymus. Thus, the early events in some cases of human acute lymphoblastic leukemia may parallel those of thymic-dependent viral murine leukemia. (7 references)

4062 CYTOLOGICAL CHARACTERIZATION AND HISTOGENESIS OF EWING'S SARCOMA. (Eng.) Takahashi, K. (Fukushima Medical Coll., Fukushima, Japan); Sato, T.; Kojima, M. *Acta Pathol Jpn* 26(2): 167-190; 1976.

To elucidate the histogenesis and nature of Ewing's sarcoma, light microscopic and histochemical investigations of primary and metastatic lesions in 27 patients were undertaken, and biopsy materials from the primary bone lesions of 7 patients were examined histochemically, enzyme cytochemically and electron microscopically. Ultrastructural observation revealed that besides intracytoplasmic and extracellular deposition of glycogen, the tumor cells possessed several cytological features characterized by intracytoplasmic microfilaments of varying thickness up to 80 A, occasional appearance of dense patches, fat droplets, desmosomal connections, and intracytoplasmic filaments often attached to the cell membrane. The tumor cells were mostly round, oval, or polygonal, but spindle-shaped or elongated cells were intermingled and occasionally contained well-developed rough endoplasmic reticulum resembling pericytes or fibroblasts. Varying amounts of diatase-resistant, PAS-positive materials were present in the intercellular spaces of the tumor; their staining behavior indicated that these materials were composed mainly of acid mucopolysaccharides and glycoproteins. The tumor cells exhibited both alkaline phosphatase and ATPase activity, but tests for nonspecific esterase activity were negative. These findings suggest that Ewing's sarcoma represents the malignant transformation of cells transitional between pericytes and vascular smooth muscle cells.

4063 ULTRASTRUCTURE AND TYPE OF EMBRYONIC RHABDOMYOSARCOMA PATHOGENESIS. (Ger.) Kastendieck, H. (Institut fuer Pathologie, Universitaet Hamburg, Martinistrasse 52, D-2000 Hamburg 20, West Germany); Boecker, W.; Huesselmann, H. *Z Krebsforsch* 86(1): 55-68; 1976.

An embryonic nasopharyngeal rhabdomyosarcoma--especially the cytoplasmic filament system--was studied by light and electron microscopy. A 10-yr-old boy with a history of therapy-resistant upper respiratory infection presented with conduction deafness, respiratory distress, and difficulty in swallowing. A seemingly encapsulated polypoid mass was found in the soft palate and adjoining nasopharyngeal surfaces, as far as the epipharyngeal wall. There was no bone infiltration. Upon light microscopy, predominantly small, undifferentiated, acidophilic tumor cells with large nuclei, dense nuclear membranes, large nucleoli, and a prominent cytoplasm were found. Multinucleate giant cells were absent; mitoses were frequent. The tumor had infiltrated and possessed no true capsule. Mucosal inflammation was acute, involving both the submucosa and juxtaposed musculature, with epithelial leucoplakia and metaplasia. There were signs of both myoblast fiber regeneration and degenerative fibrosis in transverse muscle fiber sections. Four cell types were identified electron microscopically: (a) undifferentiated mesenchymal cells with a big loosely packed nucleus and a small cytoplasmic rim with only a few cell organelles; (b) undifferentiated tumor cells with a broad cytoplasmic body that contains a dense network of nonspecific intermediate filaments with a diameter of about 100 A; (c) immature rhabdomyoblasts with randomly oriented specific myofilaments; and (d) fully differentiated rhabdomyoblasts with well-developed myofibrils often showing a sarcomeric pattern. Glycogen deposits that were seen in great masses in many tumor cells were thought to result from degenerative processes within the tumor. The cellular stages in the development of rhabdomyoblasts are basically identical to those in the embryogenesis and regeneration of striated muscle. From these observations, two developmental pathways are suggested: (a) origin of the tumor from an undifferentiated mesenchymal cell; (b) atypical regeneration of striated muscle that terminates in malignant progressive tumor growth. At present, the information about rhabdomyosarcomas supports the first; nevertheless, the second theory cannot be totally excluded. (31 references)

4064 MALIGNANT LYMPHOMA OF THE TESTIS. (Eng.) Gowing, N. F. C. *In: Pathology of the Testis*. Edited by R. C. B. Pugh. (Oxford: Blackwell Scientific Publications): pp. 334-355; 1976.

The incidence, pathology, and clinical features of malignant lymphoma of the testicle are reviewed. Lymphoreticular neoplasia accounted for 140 of 2,106 testicular tumors. Five were plasma cell tumors, and in seven, testicular involvement was found incidentally at autopsy of patients with generalized malignant lymphomas. Of the remaining 128, 8 had evidence of lymphoma elsewhere before testicular swelling was noted, but in all 128 the testicular tumor was a prominent manifestation. The peak age incidence was in the seventh and eighth decades, and only three patients were 12 yr or younger. Increasing swelling of the testis was the most common presenting symptom. Initial swelling was right-sided in 71, left-sided in 47, and simultaneously bilateral in 6. Twenty-one patients eventually had bilateral involvement in addition to the six. In 13 there was clinical evidence of disseminated lymphoma at the time of orchiectomy. There was no strong indication that trauma or cryptorchidism was etiologically involved, and there was only one case of familial incidence. Within 2 yr of orchiectomy, 62% of the patients were dead, and only 15 survived 5 yr or more. The macroscopic and histologic findings are described. The differential diagnosis includes seminoma, embryonal rhabdomyosarcoma, and pseudolymphoma. Dissemination may be very widespread, especially in fatal cases. Only three patients developed a leukemic blood picture. Among those over 50, malignant lymphoma is the second most common tumor of the testis (26%), with seminomas accounting for 37.9% in this age group, and above age 75, lymphoma is the most common testicular tumor. The long survival of a few patients after orchiectomy suggests that some malignant lymphomas are truly primary in the testis. (36 references)

4065 TERATOMA. (Eng.) Pugh, R. C. B.; Cameron, K. M. *In: Pathology of the Testis*. Pugh, R. C. B., ed. (Oxford: Blackwell Scientific Publications): pp. 199-244; 1976.

The histogenesis, classification, and clinical, pathologic, and therapeutic features of 569 cases of teratoma of the testis are reviewed. There were 28 teratoma differentiated, 312 malignant teratoma intermediate, 208 malignant teratoma undifferentiated, and 21 malignant teratoma trophoblastic. There were 4 bilateral tumors, and 303/569 affected the right side. The age range at orchiectomy was 3 mo to 76 yr, with a peak in the third decade. A history of cryptorchidism was present in 34. The presenting clinical feature was usually swelling (419) or some other testicular symptom. The duration of symptoms was variable, but in 450 it was less than 1 yr. Treatment consisted of orchiectomy with ligation of the permatic cord and postoperative radiotherapy in most cases. Retroperitoneal lymph node dissection was reserved for selected cases that did not respond to radiotherapy, and chemotherapy was reserved for advanced disease. The histopathology of each tumor type is discussed in detail. The overall 3-, 5-, and 10-yr survival rates were 47%, 45%, and 43%, respectively. Factors affecting prognosis were histologic structure, the presence of metastatic disease at the time of orchiectomy, local spread of tumor, and the amount of trophoblastic tissue in trophoblastic tumors. Children survived almost twice as well as men, as did those with histories up to 6 wk compared with those with longer histories. Cryptorchidism, testicular size, and Leydig cell hyperplasia were not correlated with survival. Alpha-fetoprotein was of no diagnostic value, but it may have use as a therapeutic monitor. (50 references)

4066 TOTIPOTENCY AND NORMAL DIFFERENTIATION OF SINGLE TERATOCARCINOMA CELLS CLONED BY INJECTION INTO BLASTOCYSTS. (Eng.) Illmensee, K. (Inst. Cancer Res., Fox Chase, Philadelphia, PA 19111); Mintz*, B. *Proc Natl Acad Sci USA* 73(2): 549-553; 1976.

A test for developmental totipotency of mouse malignant teratocarcinoma cells was conducted by cloning singly injected cells in genetically marked blastocysts. Totipotency was shown in an adult mosaic female. Clonally propagated cell lineage had differentiated in numerous normal directions. The test cells were from ''cores'' of embryoid bodies of a euploid, chromosomally male (X/Y), ascites tumor grown only in vivo by transplantation for 8 years. Cells from this transplant line provide material suitable for projected somatic and germ-line genetic analyses of mammalian differentiation based on ''cycling'' of mutation-carrying tumor cells through developing embryos. In some animals obtained from single-cell injections, tumor-derived cells were sporadically distributed in developmentally unrelated tissues. These cases can be accounted for by delayed and haphazard cellular integration, and by a marked degree of sustained cellular developmental flexibility in early mammalian development, irrespective of certain classical ''germ-layer'' designations. All mosaic mice obtained have been free of teratomas. In one case, the injected stem cell contributed only to the pancreas and gave rise to a malignancy resembling pancreatic adenocarcinoma. The high modal frequency of euploidy in these individually tested cells suggests that a near-normal chromosome complement is sufficient for total restoration of orderly gene expression in a normal embryonic environment; it may also be necessary for teratoma stem-cell proliferation to be terminated there.

4067 SEMINOMA. (Eng.) Thackray, A. C.; Crane, W. A. J. *In: Pathology of the Testis.* Edited by R. C. B. Pugh. (Oxford: Blackwell Scientific Publications): pp. 164-198; 1976.

The histogenesis, etiology, pathology, clinical aspects, and treatment of seminoma are reviewed. The incidence of this tumor in a series of 2,739 testicular tumors was 39.5%. The rate in the general population has recently been increasing. In 729 cases of seminoma, 78.6% occurred in the third through fifth decades. There were 376 tumors on the right side and 319 on the left; in 13, the tumor was bilateral. Testicular swelling was the most common presenting symptom (581/729), and pain was next (71 cases). There was little to suggest familial predisposition, and evidence for trauma as an etiologic factor was debatable. Maldescent had been present in 70 patients, but in 9 this was on the contralateral side. Seminomas developed in 29.8% of those with teratomas to form combined tumors. Macroscopic and microscopic appearance is discussed in detail. There were 30 cases of spermatocytic seminoma, and the pathology of this tumor is discussed separately. Of those without metastases at presentation, 57% were in Stage P1, 23% in Stage P2, and 20% in Stage P3. Of 27 patients presenting with abdominal seminoma, 15 had an undescended testis and 12 had normally sited testes. Age was not a significant prognostic factor, nor was the duration of symptoms before orchiectomy, nor size of tumor except when it was more than 4 times normal. The uncorrected 3-yr survivals for Stages P1, P2, and P3 were 93%, 92% and 80%, respectively. The 3-yr survivals with and without the presence of giant cells were 77% and 90%, but this difference was not significant, and at 15 yr there was little difference between these groups. Survival was positively correlated with the presence and degree of lymphocytic reaction in the tumor. The overall corrected 5-yr survival rates were 86.5% for typical seminoma, 100% for spermatocytic seminoma, and 58% for atypical seminoma. (18 references)

4068 COMBINED TUMOURS. (Eng.) Pugh, R. C. B. *In: Pathology of the Testis.* Edited by R. C. B. Pugh. (Oxford: Blackwell Scientific Publications): pp. 245-258; 1976.

The classification and the clinical, pathologic, and prognostic features of combined tumors of the testis, which contain both teratomatous and seminomatous components, are reviewed. Tumors are classified as differentiated teratoma and seminoma (TD+S), or combinations of S with malignant teratoma intermediate (MTI+S), malignant teratoma undifferentiated (MTU+S), or malignant teratoma trophoblastic (MTT+S). Among 259 mixed tumors 7 were TD+S, 132 were MTI+S, 107 were MTU+S, and 13 were MTT+S. Successive bilateral testicular tumors occurred in five, and there was a right:left ratio of approximately 6:4. The age range was 17-76, with a peak in the 30-34 quintennium. There was no familial incidence; 32 patients gave frequently vague histories of trauma and 11 had maldescent. Three had intraabdominal testes that became neoplastic and presented as acute abdominal emergencies; all three died within 8 mo. Testicular swelling was the most common presenting symptom. Thirty-three patients had evidence of metastatic disease at presentation. The duration of symptoms varied widely, but a history of less than 12 mo was obtained in 212/259. All were treated by simple orchiectomy, and postoperative radiotherapy was generally given. The distribution of metastases was determined in only a few patients, in whom the lung, abdominal lymph nodes, liver, and mediastinal and cervical nodes were the most common sites. In 34 the histology of metastases was compared with that of the primary. In 32/34 the metastases were entirely teratomatous, and in 2 only seminoma was found in the secondaries. The overall 3-yr corrected survival was 54%, compared to 100% for TD+S, 67% for MTI+S, 40% for MTU+S, and 15% for MTT+S. With some types of combined tumor, the corrected survival was distinctly worse than for pure seminoma. Metastatic disease at the time of orchiectomy carried an adverse effect on prognosis, as did stage of disease among those without metastasis. Age at orchiectomy, history of cryptorchidism, and Leydig cell hyperplasia in the involved testis did not correlate with survival, but a shorter clinical history (less than 4 1/2 mo) and smaller testicle (less than twice normal) favorably affected prognosis. (No references)

4069 ENDOCRINE AND GENETIC LESIONS. (Eng.)

Symington, T.; Cameron, K. M. *In: Pathology of the Testis*. Edited by R. C. B. Pugh. (Oxford: Blackwell Scientific Publications): pp. 259-303; 1976.

Endocrine and genetic lesions are classified, based on embryological concepts, into testicular and gonadal types. Testicular lesions include those of the interstitial (Leydig) cell (hyperplasia and tumor), the Sertoli cell (nodules in the cryptorchid and nodules or tumors in testicular feminization), and the mixed Sertoli cell/mesenchymal cell tumor. Gonadal aplasia and dysplasia include XY gonadal agenesis, pure gonadal dysgenesis, and gonadoblastomas. The benign interstitial cell tumor is uncommon (43 in 2,739 testicular tumors), and there was a 20:16 preference for the right side. Three patients had undescended testicles, and in five, the nontumorous testis was atrophic. Gynecomastia was the most frequent presenting symptom. Thirty-one of 43 patients were alive without evidence of recurrence, 16 for more than 5 yr and 6 for more than 10 yr. Death was attributed to interstitial tumor in four, 7 mo to 3 yr after orchiectomy. Larger Sertoli cell lesions have not been reported to metastasize, but there is a tendency for other testicular tumors, particularly seminoma, to develop in these patients. Thirty-two tumors were classified as mixed Sertoli cell-mesenchymal tumors. Twenty-four of these occurred under age 40, 7 in the first decade and 3 within the first year of life. Painless testicular swelling was the most common presenting feature, and cryptorchidism was present in only one case. Twenty-two patients (9 with and 9 without postoperative radiotherapy) were alive without evidence of recurrence for 1-17 yr, 14 for more than 5 yr. In at least seven cases the tumor appeared to have been malignant; metastatic sites included retroperitoneal and mediastinal lymph nodes, liver, lung, brain, and bone. Four cases of gonadoblastoma are included. Seminomalike areas of varying size occur in about half of these tumors, the prognosis for which is usually excellent, although seminomatous metastases have been recorded. Two cases of carcinoma of the rete testis were seen. One died of unrelated causes 2 yr after orchiectomy with a metastatic nodule found in the lung at necropsy, and the other died 5 mo after surgery with widespread dissemination. (78 references)

4070 YOLK-SAC TUMOUR ('ORCHIOBLASTOMA') AND OTHER TESTICULAR TUMOURS OF CHILDHOOD.

(Eng.) Brown, N. J. *In: Pathology of the Testis*. Edited by R. C. B. Pugh. (Oxford: Blackwell Scientific Publications): pp. 356-370; 1976.

A review of 130 testicular and paratesticular tumors in boys up to the age of 15, constituting 4.4% of a series of 2,941 testicular tumors, is presented. There were 53 yolk-sac tumors ("orchioblastoma"), 29 paratesticular sarcomas, 21 differentiated teratomas, 6 intermediate malignant teratomas, 7 Sertoli-mesenchyme tumors, 4 each seminoma and malignant lymphoma, and 1 each malignant teratoma undifferentiated, fibroma of the testis, cystadenoma of the epididymis, metastatic neuroblastoma, pigmented retinoblastic tumor, and tumor of the dysgenetic testis. The concept of an origin of yolk-sac tumor from the yolk sac is based largely on the architecture of the tumor. It is now being diagnosed with greater frequency. One was found in a stillborn fetus of 39 wk gestation and another noted at birth; the remainder were diagnosed at 2 mo to 5 yr. All tumors were unilateral; associated trauma was reported in one case and undescended testicle in none. The tumor grows rapidly, the mean duration of history being 3 mo. All cases were treated by orchiectomy, and one patient received immediate postoperative radiotherapy. Later metastases were treated by radiotherapy alone in two and radiotherapy combined with chemotherapy in five. Of the 53 cases, one was stillborn, 13 died 2-24 mo after surgery with multiple metastases, 24 were alive and well (5 for more than 10 yr, 7 for 5-10 yr), 2 were alive with metastases, 3 were lost to followup, and 10 were recent cases. The 3-yr corrected survival rate of 26 patients was 64%. Histology per se was of little prognostic value, but younger age and shorter duration of symptoms correlated with better prognosis. The other childhood testicular tumors are briefly discussed. Paratesticular sarcomas grew rapidly and tended to metastasize early and widely. Twelve of 27 were known to have died of their tumor, frequently within a few months. The majority of testicular teratomas occurring at less than 12 yr tended to behave in a benign fashion. Seminomas were similar to the adult seminoma; all were treated with radiotherapy, and three that had been followed were alive at 6, 7, and 10 yr after treatment. (28 references)

4071 PRIMARY INTRACRANIAL YOLK SAC TUMOR ORIGINATING IN THE PINEAL GLAND: IMMUNOFLUORESCENT DEMONSTRATION OF ALPHA-FETOPROTEIN SYNTHESIS.

(Jpn.) Yoshiki, T. (Dept. Pathology, Sapporo City General Hosp., Sapporo, Japan); Itoh, T.; Shirai, T.; Noro, T.; Tomino, Y.; Hamajima, I.; Takeda, T. *Jpn J Cancer Clin* 22(4): 355-361; 1976.

he case of a 20-yr-old man with a primary intracranial yolk sac tumor is presented. The primary tumor was located in the pineal region; biopsy revealed a diffuse proliferation of atypical tumor cells. A metastatic subdural tumor, extirpated from the lumbar spinal region, possessed the characteristic histological appearance of a yolk sac tumor. High levels of α-fetoprotein (AFP) in the cerebrospinal fluid indicated the tumor's intracranial origin. At autopsy, only a metastatic tumor was found in the posterior fossa. Immunofluorescence demonstrated a granular distribution of AFP in the cytoplasm of the tumor cells; the intra- and extracellular hyalin globules also contained AFP. Albumin, α_1-antitrypsin, and transferrin were present in the tumor cell cytoplasm in granular form. These findings suggest that the yolk sac tumor may synthesize several plasma proteins in addition to AFP. (19 references)

4072 MYCOSIS FUNGOIDES WITH LEUKAEMIC TRANSFORMATION (Meeting Abstract). (Eng.) Harrington, C. I. (Rupert Hallam Dept. Dermatology, Hallamshire Hosp., Sheffield, England). *Br J Dermatol* 95 (14/Suppl): 10; 1976.

4073 AN ELECTRON MICROSCOPIC STUDY OF LEUKEMIA CUTIS: TWO CASES IN SIBLINGS OF CHRONIC LYMPHATIC LEUKEMIA (Meeting Abstract). (Eng.) Niizuma, K. (Keio Univ. Sch. Medicine, Tokyo 160, Japan); Toyama, K. *Nihon Rynsho Denshi Kenbikyo Gakkai-Shi* 6(3/4): 273; 1973.

4074 CYTOGENETIC STUDY ON 22 PATIENTS WITH CHRONIC MYELOGENOUS LEUKEMIA (Meeting Abstract). (Eng.) Matsunaga, M. (Dept. Hematology, Atomic Disease Inst., Nagasaki Univ., Nagasaki, Japan); Sadamori, N.; Tomonaga, Y.; Tagawa, M.; Ichimaru, M. *Jpn J Hum Genet* 20(4): 267-268; 1976.

4075 ULTRASTRUCTURE AND CYTOCHEMISTRY OF THROMBOCYTES IN NORMAL SUBJECTS AND IN PATIENTS WITH STEM CELL LEUKEMIA. (Bul.) Dobreva, A. N. (Inst. Hematology and Blood Transfusion, Medical Acad., Bulgaria); Genova, V. *Vutr Boles* 14(3): 23-28; 1975.

4076 T CELL LEUKAEMIA IN THE RAT: THE PATHOPHYSIOLOGY. (Eng.) Dibley, M. (Pathology Dept., Univ. Sydney, New-South Wales, Australia); Dorsch, S.; Roser, B. *Pathology* 7(3): 219-235; 1975.

4077 DISTRIBUTION PATTERN OF FILAMENTS IN LEUKEMIA CELLS (Meeting Abstract). (Eng.) Felix, H. (Div. Cancer Res., Univ. Zurich, 8050 Zurich, Switzerland). *J Cell Biol* 70(2/Part 2): 213a; 1976.

4078 TRAPPING AND DESTRUCTION OF BLOOD-BORNE SYNGENEIC LEUKAEMIA CELLS IN LUNG, LIVER AND SPLEEN OF NORMAL AND LEUKAEMIC RATS. (Eng.) Sadler, T. E. (Dept. Surgery, Royal Postgraduate Medical Sch., Ducane Road, London, W12 OH8, England); Alexander, P. *Br J Cancer* 33(5): 512-520; 1976.

6481 FINE STRUCTURE OF PRIMARY INTRACRANIAL MALIGNANT LYMPHOMAS (Meeting Abstract). (Eng.) Jellinger, K. (Neurological Inst., Univ. Vienna, Austria); Sluga, E.; Slowik, F. *J Neuropathol Exp Neurol* 35(1): 100; 1976.

4080 MICROGLIOMA IN RENAL TRANSPLANT RECIPIENTS (Meeting Abstract). (Eng.) Johnson, J. R. (Renal Unit, Royal Prince Alfred Hosp., Australia); Britton, W. J. *Aust NZ J Med* 6(1): 92; 1976.

4081 LEIOMYOMATOUS TUMOURS OF THE INFANT KIDNEY (Meeting Abstract). (Eng.) Penchansky, L. (Dept. Pathology, Children's Hosp., Gallo, Argentina); Gallo, G. *J Pediatr* 89(2): 320; 1976.

4082 OBSERVATION OF ENDOMETRIOID CARCINOMA OF THE OVARY AND POSSIBLE TRANSFORMATION OF A PREEXISTING OVARIAN ENDOMETRIOSIS. (Fre.) Rochet, Y. (Clinique Gynecologique, Pavillon L, Hopital Edouard Herriot, 69003 Lyon, France); Meunier, P.; Cognat, M.; Lesbros, F. *Gynecologie* 26(5): 367-369; 1975.

4083 BANDING ANALYSIS OF HUMAN LEUKEMIC AND MALIGNANT LYMPHOMA CELL CHROMOSOMES (Meeting Abstract). (Eng.) Abe, T. (Dept. Preventive Medicine, Kyoto Prefectural Univ. Medicine, Kyoto, Japan); Morita, M.; Kawai, K.; Misawa, S.; Masuda, M. *Jpn J Hum Genet* 20(4): 250-251; 1976.

4084 ESOPHAGEAL CARCINOMA METASTATIC TO THE PATELLA: A CASE REPORT. (Eng.) Ashby, M. E. (Dept. Surgery, Div. Orthopedics, Martin Luther King, Jr., General Hosp., Los Angeles, CA); Dappen, N. *JAMA* 235(23): 2519-2520; 1976.

4085 LYMPHOMA AND IMMUNE DEFICIENCY. (Fre.) Daguillard, F. (Centre Hospitalier, Universite Laval, Quebec, Canada); Rousseau, C.; Lacourciere, Y.; Letarte, P.; Tremblay, M. *Union Med Can* 105(3): 399-402; 1976.

4086 ULTRASTRUCTURAL VARIATION OF LYMPHOID CELL NUCLEI. I. ELEMENTARY CHROMATIC FIBRILS OF L5178Y CELLS (Meeting Abstract). (Eng.) Kinjo, Y. (Tokyo Metropolitan Isotope Res. Inst., Tokyo, Japan); Watanabe, M. *J Radiat Res* 17(1): 27-28; 1976.

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4088 PAPILLOMA OF THE ESOPHAGUS (Meeting Abstract). (Eng.) Dourdourekas, D. (Div. Gastroenterology, Cook County Hosp., Chicago, IL); Krishnankutty, K.; Steigmann, F. *Gastrointest Endosc* 22(4): 223; 1976.

4089 NON-MYELINATED NEUROMAS WITHIN CENTRAL NERVOUS TISSUES (Meeting Abstract). (Eng.) Feigin, I. (New York Univ., New York, NY); Budzilovich, G. N. *J Neuropathol Exp Neurol 35(1): 119; 1976.*

4090 ELECTRON MICROSCOPIC STUDIES OF NEUROBLASTOMA IN CHILDREN (Meeting Abstract). (Eng.) Kemmotsu, H. (Natl. Children's Hosp., Japan); Akiyama, H.; Kodaira, Y.; Saeki, M.; Endo, M.; Katsumata, K. *Nihon Rynsho Denshi Kenbikyo Gakkai-Shi* 6(3/4): 386-387; 1973.

4091 SPREADING FACTORS IN OSTEOSARCOMA (Meeting Abstract). (Eng.) Ghosh, P. (Sydney, Australia); Harris, P.; Taylor, T. K. *J Bone Joint Surg* 58B(2): 260; 1975.

4092 NEUROBLASTOMA WITH HETEROCHROMIA AND HORNER SYNDROME (Letter to Editor). (Eng.) Spigelblatt, L. (Hopital Sainte-Justine, 3175 Chemin Sainte-Catherine, Montreal, P.Q., Canada); Benoit, P.; Jacob, J. L. *J Pediatr* 88(6): 1067-1068; 1976.

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4094 NEOPLASIA IN PATIENTS WITH CHRONIC RENAL FAILURE (Meeting Abstract). (Eng.) Miach, P. J. (Depts. Medicine and Surgical Pathology, Austin Hosp., Heidelberg, Victoria, Australia); Dawborn, J. K.; Xipell, J. *Aust NZ J Med* 6(1): 95-96; 1976.

4095 ELECTRON-MICROSCOPIC OBSERVATIONS OF HUMAN CUTANEOUS MALIGNANT MELANOMA (Meeting Abstract). (Eng.) Ishihara, K. (Natl. Cancer Center Hosp., Japan); Komatsu, N.; Yanagida, H. *Nihon Rynsho Denshi Kenbikyo Gakkai-Shi* 6(3/4): 262; 1973.

4096 LENTIGO-MALIGNA MELANOMA: AN ULTRASTRUCTURAL STUDY (Meeting Abstract). (Eng.) Bleehen, S. S. (Dept. Dermatology, Hallamshire Hosp., England); Hartley, L.; Senior, J. *Br J Dermatol* 95(14/Suppl): 14-15; 1976.

4097 A SCANNING ELECTRON MICROSCOPIC AND HISTOCHEMICAL STUDY ON CANCER METASTASIS (Meeting Abstract). (Eng.) Kishino, Y. (Dept. Pathology, Sch. Medicine, Tokushima Univ., Tokushima, Japan); Sumi, T. *Acta Histochem Cytochem* 9(1): 99; 1976.

4098 PATTERN OF DISTRIBUTION OF METASTATIC BONE DISEASE IN MAN (Meeting Abstract). (Eng.) Krishnamurthy, G. T. (Veterans Admin. Wadsworth Hosp. Center, Los Angeles, CA); Hiss, J. M.; Tubis, M.; Blahd, W. H. *J Nucl Med* 17(6): 526; 1976.

4099 ULTRASTRUCTURAL CHARACTERISTICS OF NORMAL PRENEOPLASTIC AND NEOPLASTIC HUMAN TRANSITIONAL EPITHELIUM (Meeting Abstract). (Eng.) Lee, M. M. L. (Depts. Anatomy, Urology and Pathology, Columbia Univ., New York, NY 10032); Tannenbaum, S.; Tannenbaum, M. *J Cell Biol* 70(2/Part 2): 357a; 1976.

4100 DIFFERENCES IN CELL SURFACE MORPHOLOGY BETWEEN MALIGNANT AND BENIGN HUMAN BREAST LESIONS ARE A FUNCTION OF SPECIMEN PREPARATION (Meeting Abstract). (Eng.) Maser, M. D. (Dept. Pathology, Creighton Univ., Omaha, NB 68178). *J Cell Biol* 70(2/Part 2): 224a; 1976.

4101 THE RELATION OF ADENOMYOSIS UTERI TO COEXISTENT ENDOMETRIAL CARCINOMA AND ENDOMETRIAL HYPERPLASIA. (Eng.) Greenwood, S. M. (Div. Lab. Medicine, Geisinger Medical Center, Danville, PA 17821). *Obstet Gynecol* 48(1): 68-72; 1976.

4102 ANALYSIS OF GRAIN COUNT DECAY PATTERNS IN HUMAN BREAST CANCER LABELED WITH TRITIATED-THYMIDINE IN VIVO (Meeting Abstract). (Eng.) Sklarew, R. J. (New York Univ. Res. Service, Goldwater Memorial Hosp., Roosevelt Island, New York, NY 10017); Hoffman, J.; Post, J. *J Cell Biol* 70(2/Part 2): 239a; 1976.

4103 GENETICS AND BREAST CANCER. (Eng.) Anderson, D. E. *In: Early Breast Cancer: Detection and Treatment.* Gallager, H. S., ed. (New York: John Wiley & Sons): pp. 41-49; 1975.

4104 MIXED MESODERMAL TUMORS OF THE OVARY. (Fre.) Hoang-Ngoc, M. (Laboratoire d'Anatomie Pathologique, C.H.U. d'Amiens, Amiens, France); de Saint-Maur, P. P.; Jagueux, M.; Gontier, F.; Smadja, A. *Gynecologie* 26(5): 357-359; 1975.

4105 FINE STRUCTURE OF TESTICULAR INTERSTITIAL CELL IN MEN WITH PROSTATIC CANCER (Meeting Abstract). (Eng.) Mori, H. (Ehime Univ. Sch. Medicine, Ehime, Japan). *J Electron Microsc (Tokyo)* 24(3): 187; 1975.

4106 ULTRASTRUCTURE OF EXPERIMENTAL COLONIC TUMOR OF RAT (Meeting Abstract). (Eng.) Kohro, T. (Univ. Niigata Sch. Medicine, Niigata City, Japan); Tazawa, K.; Soga, J.; Sano, M.; Sakashita, A.; Narisawa, T.; Soeno, T. *Nihon Rynsho Denshi Kenbikyo Gakkai-Shi* 6(3/4): 410; 1973.

4107 LATE OCCURRENCE OF PRECANCEROUS CHANGES AND CARCINOMA OF THE GASTRIC STUMP (Meeting Abstract). (Eng.) Janunger, K. -G. (Dept. Surgery and Pathology, Univ. Umea, Umea, Sweden); Bergmann, F.; Domellof, L.; Eriksson, S. *Scand J Gastroenterol* [Suppl] 10(Suppl. 34): 11; 1975.

4108 CLINICO-PATHOLOGICAL INVESTIGATION ON PATIENTS WITH EARLY GASTRIC CANCER'' AND INTERMEDIATE GASTRIC CANCER'' IN CHILE. (Eng.) Csendes, A. (Depts. Surgery, Medicine, Radiology, Public Health and Pathology, Univ. Chile, Santiago, Chile); Strauszer, T.; Doerr, E.; Fernandez, E.; Medina, E.; Velasco, N.; Smok, G.; Jara, L. *Jap J Clin Oncol* 6(1): 11-16; 1976.

4109 INTESTINAL-TYPE CARCINOMA OF GALLBLADDER: A HISTOCHEMICAL AND IMMUNOLOGIC STUDY. (Eng.) Laitio, M. (Central Hosp. Seinajoki, Seinajoki, Finland); Hakkinen, I. *Cancer* 36(5): 1668-1674; 1975.

See also:

* (Rev): 3615, 3616, 3625, 3629, 3639, 3643, 3649, 3655, 3656, 3665, 3666, 3667, 3668, 3669, 3670, 3671, 3672, 3673, 3674, 3675, 3676, 3677, 3678, 3679, 3680, 3681, 3682, 3683, 3684, 3685, 3686, 3687, 3688, 3689, 3690, 3691, 3692, 3693, 3694, 3695, 3696, 3720, 3721, 3722, 3723, 3725, 3726, 3727, 3728, 3729, 3730, 3731
* (Chem): 3739, 3749, 3754, 3761, 3772, 3786, 3807, 3810, 3817, 3820, 3828
* (Phys): 3832, 3855
* (Viral): 3909, 3914, 3923, 3939, 3954, 3961, 3982, 3985, 3996, 3998
* (Immun): 4005, 4022, 4046, 4052, 4055
* (Epid-Biom): 4110, 4112, 4114, 4117, 4118, 4119, 4124, 4126, 4130, 4132, 4138, 4139, 4142, 4143

4110 USE AND LIMITATIONS OF AUTOPSY DATA. (Eng.) Pickren, J. W. *In: Fundamental Aspects of Metastasis*. Weiss, L., ed. (Amsterdam: North-Holland Publishing Co.): pp. 377-384; 1976.

Autopsy data accumulated at the Roswell Park Memorial Institute since 1914 are available for over 7,000 cases of cancer. The autopsy protocol was designed to study the spread of cancer, and basic identification data such as the patient's age and sex, duration of the disease, histogenetic type of cancer, and typographical site are included. All of the body organs are identified, and a notation is made as to whether the organ is involved or not according to a master code. Lung cancer was studied as an example of the use of autopsy data. Cases were selected on the basis of two criteria: (a) the patient had cancer that had been considered curable by lung resection, and (b) the patient died within 1 mo of resection. These cases were then subdivided into histological types. Cases of anaplastic small cell carcinoma invariably showed distant metastasis, obviating the chance of cure by operation even if the patient had survived the immediate postoperative period. Patients with squamous cell carcinoma, on the other hand, had relatively few distant metastases, but 25% had local disease within the chest. The results suggest that patients with squamous cell carcinoma of the lung should have localized therapy to the mediastinum after surgical excision, while systemic therapy is required for anaplastic small cell carcinoma for any possible chance of cure. (5 references)

4111 SEASONALITY, NATALITY AND HERD IMMUNITY IN FELINE PANLEUKOPENIA. (Eng.) Reif, J. S. (Sch. Veterinary Medicine Univ. Pennsylvania, Philadelphia, PA 19174). *Am J Epidemiol* 103(1): 81-87; 1976.

The seasonal distribution of feline panleukopenia (FPL) was examined. FPL was diagnosed in 185 of 7043 feline admissions (2.63%) at a university veterinary hospital over an 8-yr period. FPL was found to have a distinct seasonal pattern with peaks occurring during July, August, and September. Seasonal peaks were noted in all the years studied. Cats less than 1 yr of age accounted for 70% of the total morbidity. Analysis of 47,786 purebred litters born during 1970-1972 revealed a peak number of births during April, May and June with a national median of May 29. A unifying hypothesis is presented to account for the seasonal occurrence of FPL. An influx of susceptible cats occurs annually following the birth of large numbers of kittens each spring, and disappearance of maternal immunity during the next 2-3 mo. The addition of a large number of susceptible kittens leads to the development of summertime epidemics and serves to exemplify the principles of ''herd immunity''.

4112 CANCER STATISTICS, 1976. A COMPARISON OF WHITE AND BLACK POPULATIONS. (Eng.) Seidman, H. (American Cancer Society, New York, NY); Silverberg, E.; Holleb, A. I. *CA* 26(1): 2-29; 1976.

White and black US populations are compared with regard to cancer statistics. Nonmelanoma skin cancer and all in situ carcinomas are excluded from consideration. The lifetime probability of developing cancer is about 1/4, ranging from 24% in black to 31% in white females, with white and black males at 29% and 27%, respectively. The age-adjusted incidence of cancer per 100,000 in males has increased between 1947 and 1969, rising only slightly in whites but very markedly in blacks, largely due to increases in cancers of the lung, colon-rectum, prostate, and esophagus. Both white and black females showed decreases in age-adjusted cancer incidence. Five-year survival rates in males were 31% in whites and 21% in blacks; in females, the rates were 47% in whites and 37% in blacks. Distribution by cancer site indicates that incidence of cancer of the esophagus has declined in whites, while rapidly rising in blacks. Stomach cancer has substantially declined in all segments of the population. Colon and rectal cancer incidence rates have changed little in whites, but appreciably in blacks. Lung cancer incidence rates rose sharply in both white males and black males, the latter now exceeding the former. The incidence of breast and ovarian cancer is higher in white females, while the incidence of cancer of the cervix uteri is higher in blacks. The incidence of prostate cancer is much higher for blacks than whites and urinary bladder cancer is higher in whites than blacks, but in both instances, fewer blacks are diagnosed in a localized stage and their survival rates are lower. The kidney is the only cancer site that has lower survival rates in whites than blacks. (No references)

4113 SOME EPIDEMIOLOGICAL ASPECTS OF MAXILLOFACIAL MALIGNANT TUMORS IN SERBIA. (Cro.) Skokljev, A. (Klinika za Maksilofacijalnu i Oralnu Hirurgiju, VMA - Vojnomedicinska Akademija, Beograd, Jugoslavia); Merkas, Z.; Piscevic, A.; Ristic, J.; Trbojevic, P.; Roncevic, R. *Zbornik Vojnomed Akad* 44-48; 1973.

The clinical data was analyzed on 3,456 patients hospitalized with malignant maxillofacial tumors during 1959-1969. The morbidity was found to be 0.045% and was greatest (0.089%) in northeast Serbia, especially in the district of Smederevo (0.10%). The tumors were 2.5 times more frequent in men than women, and they were found predominantly among farmers. The etiological factors were identified as tobacco in 43 cases, trauma in 30, precancerosis in 13, syphilis in 5, and alcohol in 4 cases. In one-third of the cases the tumors were easily detected, and an early diagnosis (during the first 3 mo after the manifestation of the first symptoms) was made. Tumors of the oral cavity amounted to 77.8%, followed by tumors of the upper jaw and the tongue. Morphologically, flat-cell (squamous cell) carcinomas were most frequent (73.5%).

4114 STATISTICAL ANALYSIS OF TUMOR PATIENTS HOSPITALIZED IN TUNISIA IN 1971. (Fre.) Souissi, H. (No affiliation given). *Tunis Med* 53(2): 127-135; 1975.

This is a report of a study of tumors in Tunisia in 1971, through a review of hospital admissions and their outcome in release or death. Tumors, 4,168 in all, held the 11th rank among causes for hospitalization. Among the 2,894 malignant types, 808 (28%) concerned the digestive tract, notably the liver and biliary ducts (166), stomach (165), and mouth and pharynx (127). The 582 tumors of the respiratory system (20%) mainly affected the larynx (154) and the lungs (268). All other locations accounted for the remaining 1,504 tumors (52%), with breast cancers in the first place (272), followed by Hodgkin's disease (216), leukemia (192), cancers of the uterine cervix (136), skin (125), and nervous system (100). Benign tumors, totaling 1,274, most often involved the skin (307) or the genitourinary organs (178). For all causes of hospitalization, the mortality rate reached 2.4%; for tumors, it was 5.11%. Tumors ranked as the eighth most common cause of death. For each 1,000 hospitalizations, there were 67 deaths of malignant tumors, 14 of benign tumors. Of the digestive tract cancers, the most lethal were those of the esophagus (22 deaths per 100 hospitalizations), intestine (18/100), and pancreas (17/100). The death rate seemed generally low for the respiratory tract (4 to 5/100). For all other locations, the highest mortality occurred in tumors of the nervous system (31/100) and leukemia (16/100). It was low for tumors of the skin, breast, and uterine cervix. The lethality of benign tumors in the genitourinary organs slightly exceeded that of malignant tumors, 2.8 against 2.4. Indigent cases (60% of hospitalizations) accounted for over 75% of cancers of the digestive tract. Members of other social groups were more frequently victims of Hodgkin's disease or breast and larynx tumors. There were no group differences for benign tumors. Tumors became more frequent after age 30 and kept increasing with advancing age. Among the young, Hodgkin's disease was first. Males accounted for 51% of malignant tumors and females for 42%, despite the existence of cancers specific to women. Tumors of the respiratory system were the most common in men (at a rate quadruple that of women), followed by Hodgkin's disease and leukemia. Tumors of the breast, uterine cervix, and uterus were the most prevalent in women.

4115 CERVICAL CANCER IN YUGOSLAVIA: A NATIONWIDE STUDY OF EPIDEMIOLOGICAL FACTORS.

(Eng.) Kulcar, Z.; Kessler, I.; Grgurevic, M.; Zimolo, A. *In: Host Environment Interactions in the Etiology of Cancer in Man. Proceedings of a Meeting Held at*
of Cancer in Man. Proceedings of a eeting eld at
Primosten, Yugoslavia, 27 August-2 September 1972. International Agency for Res. on Cancer (Lyon, France): pp. 57-62; 1973.

A field study of cervical cancer in Yugoslavia is described and some preliminary results are given. One group of patients was selected from a society in which male circumcision is practiced and another from a society not practicing circumcision. Matched controls were selected from hospitalized women without venereal disease or cervical cancer. The information collected included the following: general medical and anthropometric data; socioeconomic characteristics; family history; and menstrual, obstetric, marital, and sexual history. All hospital records were abstracted, and a complete gynecological examination was performed. Histological specimens were reviewed for diagnostic accuracy. Finally, blood samples were drawn and centrifuged and serum specimens analyzed for antibodies to genital herpesvirus. The following preliminary conclusions were drawn: patients from the circumcised populations had more pregnancies than other patients, but figures did not differ from controls; partners of cancer patients tended to be more promiscuous than those of controls, regardless of circumcision status; patients from the circumcised population tended to wash themselves at menses more often than those from the uncircumcised population, similar to controls; cases from both populations tended to have their first intercourse earlier than controls; cases from both populations moved and migrated as often as controls; and the prevalence of antibodies to genital herpesvirus was greater among cases from both populations than among controls. (2 references)

4116 SCREENING FOR CARCINOMA OF THE CERVIX.

(Eng.) Anonymous (No affiliation given). *CMA J* 114: 1013-1026; 1976.

Medical screening programs for carcinoma of the cervix are being conducted to detect disease at an early, more treatable stage in asymptomatic women. Descriptions of screening programs in various provinces of Canada point out the difficulties in obtaining true baseline data for the population and obtaining wide participation by the majority of women. Those considered at risk for cervical carcinoma and who should be included in the program include all sexually active women over 18 yr. An initial smear at 18 yr followed by a repeat smear 1 yr later is recommended to allow for the possibility of false-negative sampling; thereafter, smears at 3-yr intervals until age 35 and at 5-yr intervals until age 60 are recommended. If all smears have shown no significant atypia, the woman may be dropped from the program at age 60. Those considered at such a low risk that they need not be included are women with no sexual activity at all, women over 60 yr who have had regular smears with no atypical cytology, and women with hysterectomy for benign disease with complete removal of the cervical epithelium. Analysis of a screening program conducted in Canada in 1966 showed that it has had a significant effect in reducing mortality from carcinoma of the uterus.

4117 CERVICAL CANCER SCREENING PROGRAMS. I. EPIDEMIOLOGY AND NATURAL HISTORY OF CARCINOMA OF THE CERVIX.

(Eng.) Gellman, D. D. (Health and Welfare Canada, Ottawa ON K1A 1B4, Canada). *CMA J* 114(1): 1003-1004, 1007-1012; 1976.

Medical screening programs for cervical carcinoma also detect dysplasia and carcinoma in situ in early stages. Mean age at time of diagnosis for carcinoma in situ is 34 yr, for carcinoma in situ with microinvasion 43.7 yr, for occult carcinoma of cervix 48.6 yr, and for clinical carcinoma of cervix 52 yr.

Prospective studies have shown an annual progression rate from dysplasia to cancer of 5% to 6.4%. In some patients the in situ phase might be skipped or not detectable and invasive cancer might develop directly from dysplasia. Cytologic followups with cervical biopsies have shown regression rates for dysplasia of 30%-40%; the apparent therapeutic effect of the biopsies must be acknowledged. Reported regression rates for carcinoma range from 0 to 25%, with the lowest regression rates in patients followed by cytology rather than by biopsy; this suggests a possible therapeutic effect of the biopsies. Clinical experience indicates that it is extremely rare for invasive carcinoma to develop without carcinoma in situ being present initially. The natural history of carcinoma of the cervix with its precursor stages indicates that this disease is especially well-suited for medical screening programs. (41 references)

4118 HUMAN CERVICAL CANCER AS A VENEREAL DISEASE. (Eng.) Kessler, I. I. (Dept. Epidemiology, Sch. Hygiene and Public Health, The Johns Hopkins Univ., Baltimore, MD 21205). *Cancer Res* 36(2): 783-791; 1976.

Evidence on the venereal nature of HSV-2 infections in man was reviewed in the light of existing knowledge on other, more established, venereal diseases. The operation of venereal factors in human cervical cancer was suggested by demographic and epidemiological data. Recent evidence supporting the genital herpesvirus hypothesis is consistent with this notion. A controlled epidemiological investigation was undertaken on the role of the male coital partner in cervical carcinogenesis to determine if the risk of developing cervical cancer increased among the wives of men who at some other time were married to other women who developed cervical cancer. With one-quarter of the study completed, a total of 14 marital clusters were identified in which two or more wives of the same men developed cervical carcinoma. The expected number of such clusters was estimated to be four.

4119 GENETIC FACTORS IN BREAST CANCER. (Eng.) Lynch, H. T.; Guirgis, H.; Lynch, J.; Kraft, C.; Thomas, R. *In: Cancer Genetics.* Edited by H. T. Lynch. (Springfield, IL: Charles C. Thomas): pp. 389-423; 1976.

Particular familial contexts of breast cancer, with specific patterns of tumor associations, are becoming clearer with careful investigation. A study currently in progress concerns a large kindred manifesting, over four generations, the syndrome of breast cancer, sarcoma, leukemia, and brain tumors. Concordance-discordance analysis revealed findings consistent with an autosomal dominant pattern of genetic transmission. Breast cancers were found at an unusually young age (23, 25, and 29); brain tumors occurred in six family members. Cutaneous abnormalities consistent with diseases such as neurofibromatosis (which might explain the high occurrence of brain tumors) were not found. Several of these families are currently under more detailed investigation. It was concluded that breast cancer etiology is heterogeneous. Some cases of breast cancer appear to be sporadic, possibly due to fresh gene mutations, unknown environmental influences, and/or the interaction of these events. (69 references)

4120 RISK FACTORS IN BREAST CANCER--A PROSPECTIVE STUDY. (Eng.) Shapiro, S.; Goldberg, J.; Venet, L.; Strax, P. *In: Host Environment Interactions in the Etiology of Cancer in Man. Proceedings of a Meeting Held at Primosten, Yugoslavia, 27 August-2 September 1972.* International Agency for Res. on Cancer and John E. Fogarty International Center of the Natl. Insts. of Health USA. (Lyon, France): pp. 169-182; 1973.

The results of a prospective study of risk factors in breast cancer are presented. About 20,000 women, aged 40-64 yr, participated in the screening program and were followed for 5 yr. Elevated risks for breast cancer were observed for higher educated women, Jewish women, and unmarried women, but the margins were small, the risks among these women being only 20%-30% above contrasting groups. Increasing age at first pregnancy, and low gravidity (fewer than three pregnancies) were associated with a 50-100% elevation in relative risk. When these two characteristics were examined together, women first pregnant at an early age (under 20 yr) had a relatively low cancer risk regardless of how many pregnancies they subsequently had. Menarche at ages under 15 was associated with a 50% rise in relative risk. The elevation in relative risk for women with prior benign breast conditions was 60%-100%. Familial history of breast cancer (mothers and sisters) appeared to be related to a 30% increase in risk.

4121 THE EPIDEMIOLOGY OF OVARIAN CANCER. (Eng.) Campbell, H. *In: Gynaecological Malignancy: Clinical and Experimental Studies.* Edited by M. G. Brush and R. W. Taylor. (Baltimore: The Williams & Wilkins Co.): pp. 111-132; 1975.

A review of the epidemiology of ovarian cancer reveals that it is not a single entity but a group of separate diseases, each of which may have a distinct etiology. That is why detailed epidemiological studies have failed to develop a viable clear-cut hypothesis concerning etiology. The confusion in the terminology of the morbid pathology is indicative of the general difficulty in defining the separate types of ovarian cancers. This review also shows that ovarian cancer is the most common gynecological neoplasm, the incidence of which is still increasing, especially in single women. The incidence may be higher in professional women and in women associated with the clothing and textiles or sales and service industries. The incidence is low on the Japanese mainland, but increases sharply among Japanese migrants. There is a genetic association in some families, but the extent and cause of this has not been determined. The 5-yr survival rate is only about 30% in most areas, largely because of the late stage at which the cases are diagnosed. Smoking, radiation, and external carcinogens apparently do not play an important part in the etiology of the disease, but there is a possible correlation with talc. A viral agent may play a part, in that mumps was more frequently reported by the cases than by the controls; however, this has not been confirmed.

4122 EPIDEMIOLOGY OF PROSTATIC CANCER. (Eng.) Hutchinson, G. B. (Harvard Sch. Public Health, 665 Huntington Ave., Boston, MA 02115). *Semin Oncol* 3(2): 151-159; 1976.

The incidence of prostate cancer is second only to that of lung cancer in US men. The rate increases with age (from 50-80) and has increased with time since 1937. The incidence in nonwhites is higher and rising at a steeper rate than in white men, and, in general, mortality rates parallel incidence rates. Although many carcinogenic factors may be involved, the data suggest that one or more has changed in intensity over the past 30 yr. The white-nonwhite difference has not been explained, but the data suggest the presence of a carcinogen more common to nonwhites. There is evidence, based on geographic variation of incidence and migration studies, that environmental factors rather than genetic factors account for a substantial part of the international variations. It seems that latent carcinoma progresses to invasive carcinoma slowly if at all and that benign hyperplastic prostate is not the histologic precursor of prostate cancer. Marital and sexual factors are reviewed and it is concluded that hormonal factors are involved in the etiology. Socioeconomic status, religion, and urban residence do not seem to play major roles. The surgical treatment of prostate hyperplasia as a preventive measure is empirical, since hyperplasia may not be a precursor of cancer; however, at present it seems to be the treatment of choice for hyperplasia as well as for the carcinoma. (32 references)

4123 ON THE EPIDEMIOLOGY OF GASTRIC CANCER. (Ger.) Wolff, G. (Zentralinstitut fuer Krebsforschung, Akademie der Wissenschaften der DDR--Robert-Rossle-Klinik, DDR--1115 Berlin-Buch, Lindenberger Weg 80, East Germany); Laeuter, J. *Arch Geschwulstforsch* 46(1): 1-14; 1976.

A control group of 766 patients without conspicuous gastric disease and 257 surgical patients with histologically confirmed gastric carcinoma were interviewed with a standardized questionnaire for 32 variables. The two groups were compared by multivariate variance analysis. A univariate analysis of three other variables (blood groups, and tobacco habituation and type) is included. There were twice as many males as females in the cancer group and slightly more males among the controls; all patients were over 50 yr of age. The eight variables significantly linked with gastric cancer are listed in diminishing order: lower food intolerance (no food allergies), higher alcohol consumption, residential site (proximity to a metropolitan area), occupation (unskilled worker), early loss of teeth or longer use of dental prostheses, vehicular driving (private or occupational), absence of gallstones, and longer use of tobacco. Twelve other variables discriminated less significantly between control and cancer groups. Additional analyses matched 267 patients with normal gastric mucosa or negligible gastritis and 499 patients with chronic atrophic gastritis and the 257 cases of gastric carcinoma. The variable responses were nearly identical with those of the parent study. A special study comparing 81 females with confirmed stomach cancer and 367 random female controls followed the same pattern; in addition, a history of miscarriage was significantly linked to increased incidence of gastric carcinoma. The univariate assays showed a slightly higher incidence of blood group O and a slightly lower incidence of blood group A in stomach cancer, neither of which were significant. Smoking of cigars and/or cigarettes was significantly correlated with gastric cancer, and a higher lung cancer morbidity was found among all smokers. Direct contact between the gastric mucosa and exogenous carcinogens is implicated as the most important source of stomach cancer. (44 references)

4124 TREATMENT OF GASTRIC CANCER. (Eng.) Cady, B. (Dept. Surgery, Lahey Clinic Foundation, 605 Commonwealth Ave., Boston, MA 02215); Ramsden, D. A.; Choe, D. S. *Surg Clin North Am* 56(3): 599-605; 1976.

The etiology, incidence, detection, and treatment of gastric cancer is discussed. Evidence indicates that diet is related most closely to the cause and incidence of the disease. The most important aspect of diagnosis is a high index of clinical suspicion in patients with gastrointestinal symptoms and unexplained anemia. Any patient with a gastric ulcer cannot be considered cancer-free until confirmed by fiberoptic endoscopy and direct biopsy. Low serum pepsin levels and hypoacidity as well as high serum gastrin levels may be useful in detecting high-risk patients. Serologic immunoassay may be a future diagnostic tool for gastric cancer. Surgery is by far the most common treatment; however, fair results are sometimes achieved with aggressive multinodality therapy. The use of highly aggressive resections is discouraged because of the poor survival and cure rates. If curative resections are not feasible, palliative resections can be helpful in improving the quality of life in some instances. Data are presented that demonstrate that radical removal of the lymph nodes does not increase the success rate. New combined treatments are urgently needed for gastric cancer therapy. (15 references)

4125 MALIGNANT NEOPLASMS OF THE STOMACH IN BERLIN, CAPITAL OF THE G.D.R.--AN ANALYSIS OF THE STATE OF DIAGNOSIS AND TREATMENT. (Ger.) Berndt, H. (Zentralinstitut fuer Geschwulstforschung der AdW, DDR-1115 Berlin-Buch, Lindenberger Weg 80, East Germany); Dietrich, D.; Gregor, M.; Hansche, G.; Hansche, R.; Witt, K. *Arch Geschwulstforsch* 46(1): 15-23; 1976.

A review of 7,386 new registrations of stomach cancer in Berlin from 1955 through 1969 and for the year 1973 is presented. Although the number of aging females in the area is atypically high, the sex ratio of 52 males to 48 females remained relatively constant. Lymphatic tumors, tumors of the cardia, and those the diagnoses of which were doubtful were excluded. Tumors undiagnosed clinically but subsequently found at autopsy were included. The percentage of both sexes over age 65 increased from 61.4% in 1955-9 to 73.4% in 1973. The male incidence per 100,000 male inhabitants was 55 for 1955-9, 43.7 for 1960-4, and 48.9 for 1965-9; the respective female incidence was 36.6, 29.8, and 38.0. The lower morbidity of 1960-4 might reflect indifferent clinical reporting. Approximately 9% of the cases were in stage I, 21% in stage II, 33% in stage III, and 37% in stage IV for 1955-69. In 1973, when improved diagnostic procedures were routine, the percentage of cases in operable stages I and II was paradoxically lower (and that in stages III and IV higher) than in earlier years. These data might reflect the demographic composition of an aging urban population. There was no unequivocal relation between the duration of complaint and diagnosis of malignant stage. For stages I and II approximately 15% were treated earlier than 1 mo, 32% were treated between 1 and 3 mo, and 25% between 3 and 6 mo after the onset of symptoms. Only patient age and malignant stage are prognostically valuable in a review of the patient population between 1955 and 1965. The survival rate in patients 65 yr or older is about 7%; the rate for patients up to 65 yr is 30%-35%. The survival rate for stages I and II varied between 31% and 46%; the rate for stages III and IV, between 4.7% and 5.7%. There is some evidence that stage I and II (but not III or IV) survival rates improved between 1955 and 1965, although it is contrary to the meager statistics from other urban areas. (13 references)

4126 MORBIDITY OF GASTRIC CANCER AND OTHER MALIGNANT NEOPLASIAS IN SUBJECTS HAVING BEEN GASTRECTOMIZED FOR ULCER.

(Rus.) Patiutko, Iu. I. (Inst. Experimental and Clinical Oncology, Acad. Medical Sciences, USSR, Moscow, USSR). *Vestn Akad Med Nauk SSSR* (2): 88-92; 1976.

The morbidity of 942 patients followed for 22-28 yr after gastrectomy for ulcers was determined. Malignancies caused death in 87/274 who died at various postoperative stages. Essential differences between anticipated and actual tumor locations were found only for stomach cancer and lung cancer in men. Fifteen died from stomach cancer, but the anticipated figure was 29.2. Lung cancer developed in 30 men, twice the anticipated number among patients gastrectomized for stomach ulcer, but anticipated and actual figures did not differ for those gastrectomized for duodenal ulcer. The longer the period of time following gastrectomy, the greater the incidence of stomach cancer. Among patients gastrectomized before age 50, the actual number of cases of stomach cancer significantly exceeded the number anticipated; anticipated and actual cases were the same for patients gastrectomized at 50 yr or later.

4127 NATIONAL MARINE FISHERIES SERVICE PRELIMINARY SURVEY OF SELECTED SEAFOODS FOR MERCURY, LEAD, CADMIUM, CHROMIUM, AND ARSENIC CONTENT.

(Eng.) Zook, E. G. (Southeast Utilization Res. Center, Natl. Marine Fisheries Service, Natl. Oceanic & Atmospheric Admin., U.S. Dept. Commerce, College Park, MD 20740); Powell, J. J.; Hackley, B. M.; Emerson, J. A.; Brooker, J. R.; Knobl, G. M., Jr. *J Agric Food Chem* 24(1): 47-53; 1976.

A preliminary screening study was conducted on mercury, lead, cadmium, chromium, and arsenic contents of 34 commonly consumed seafoods. A total of 334 samples were analyzed and, in general, 10 samples per species were obtained from a location. More than 96% of the samples fell below the Food and Drug Administration guideline of 0.5 ppm for mercury, with a mean level of 0.13 ppm. Mean lead values among species tended to be rather uniform, and only a few species averaged higher than 0.6 ppm. No species mean exceeded 1 ppm. Cadmium content of most seafoods averaged less than 0.2 ppm. Chromium was present at less than 0.4 ppm in all species. Arsenic levels showed the widest variation and were higher for some species than the other elements tested. Some possible relationships between elements within each species were encountered, but the data were too limited to draw firm conclusions.

4128 EXPERIENCE WITH ASBESTOS DISEASE AND ITS CONTROL IN GREAT BRITAIN'S NAVAL DOCKYARDS.

(Eng.) Harries, P. G. (Medical Res. Unit, No. 1 East Ave., H. M. Naval Base, Devonport, England PL1 4RU). *Environ Res* 11: 261-267; 1976.

Although improved codes of practice and a reduction in the use of asbestos in shipbuilding will reduce the incidence of asbestos-related disease in the future, new cases are still being seen due to the long period between exposure and disease. At one facility in Plymouth, 156 men were awarded disability pensions for asbestosis in the 10 yr up to 1973. Laggers and sprayers had the highest proportion, but other occupations were also seen because of their close proximity to asbestos workers. Radiographic investigation of 457 workers with pleural abnormalities revealed calcified plaques in 103 men, uncalcified plaques in 285, and diffuse fibrosis in 69. The majority of these men worked near asbestos laggers, sprayers, or strippers. There were 55 deaths at this facility from mesothelial tumors in the 10 yr up to September 1973, only 2 of which were asbestos workers.

4129 PREDICTIVE VALUE OF CARCINOGENESIS BIOASSAYS.

(Eng.) Maltoni, C. (Inst. Oncology and Tumour Centre, Bologna, Italy). *Ann NY Acad Sci* 271: 431-443; 1976.

Bioassays provide an important means of predicting the oncogenic risk of particular occupational and environmental agents to man. In fact, the three most important occurrences of environmental and occupational tumors discovered after 1970 were directly or indirectly predicted by animal studies. The first was the adolescent clear-cell vaginal adenocarcinoma found in girls born to mothers treated during pregnancy with synthetic nonsteroid estrogen therapy. As long ago as 1938, it was demonstrated that stilbestrol had carcinogenic properties, with mammary tumors arising in male mice treated with the compound;

several years later, the same hormone was shown to produce a variety of tumors among different animal species. The second occurrence was that of pulmonary carcinoma among workers exposed to bis(chloromethyl) ether, which had previously been shown to produce sc fibrosarcomas and skin carcinomas when injected sc into rats or applied topically to mice. When the compound was inhaled by rats, it induced squamous cell carcinomas of the lung. The third example was provided by the identification of liver angiosarcomas in workers occupationally exposed to vinyl chloride (VC), after the carcinogenicity of the compound had been demonstrated in rats. Data from inhalation experiments with VC in rats, mice, and hamsters and from ingestion experiments in rats are reported in detail. Sixty Swiss mice of both sexes were treated by inhalation of VC in air at 10,000 ppm for 4 hr/day, 5 days weekly for 30 wk. After 81 wk 55 developed pulmonary tumors, 13 had mammary carcinomas, 8 had liver angiosarcomas, 9 had vascular tumors, and 3 had epithelial tumors of the skin; of 150 untreated mice, 8 developed pulmonary tumors and 1 a vascular tumor after the same length of time. Bioassays can also be used to predict the carcinogenicity of inorganic substances. Various inorganic materials were tested by sc injection of 30-mg quantities into Sprague-Dawley rats. Of 40 animals in each group, 9 developed rhabdomyosarcomas and fibrosarcomas with neochromium, 8 with chromium allumen, 26 with chromium yellow, 27 with molybdenum orange, 16 with cadmium yellow, and 1 with iron yellow, after 125-150 wk. None of 140 control animals developed neoplasms. Of 49 male and 48 female Sprague-Dawley rats, 34 and 31 developed peritoneal mesotheliomas following the endoperitoneal injection of 25 mg of crocidolite. (14 references)

4130 LEUKEMIA ASSOCIATED WITH BENZENE EXPOSURE.

(Eng.) Vigliani, E. C. (Clinica del "Luigi Devoto," Univ. Milan, Milan, Italy). *Ann NY Acad Sci* 271: 143-151; 1976.

Clinical and epidemiological data indicate that benzene has a strong leukemogenic action in man; the leukemias produced are mostly acute and myeloblastic in type, occurring directly or following aplastic changes in the bone marrow. Of 66 patients diagnosed as having chronic benzene poisoning, patients with less than 3 million RBC and less than 4,000 WBC (mostly workers from rotogravure plants, shoe factories, and other industries using benzene as a solvent), there were 18 deaths. Of these, 7 died from aplastic anemia and 11 from leukemia. Very often leukemia develops in subjects with benzene-induced hyporegenerative anemia or pancytopenia of fairly long standing and constitutes the acute terminal stage of the disease. The leukemia might become clinically apparent only a few weeks before death; in these cases the anemia can be considered a preleukemic stage. Aplastic anemia generally occurs in subjects while they are still exposed to high concentrations of benzene; leukemia may occur at the same time, or fairly shortly after cessation of exposure. In a few cases there may be a long latent period between the end of work with benzene and the onset of leukemia. The risk of acute leukemia among workers heavily exposed to benzene (from 200 to 500 ppm) in the rotogravure and shoe industries of the provinces of Milano and Pavia, Italy, was at least 20 times higher than that for the general population. A number of case histories of benzene leukemia are outlined. (16 references)

4131 ROLE OF OCCUPATION IN THE OCCURRENCE OF CANCER OF THE LARYNX.

(Ser.) Spalajkovic, M. (Med. Faculty Univ., Belgrade, Yugoslavia); Mitrovic, K. *Med Pregl* 28(5-6): 245-247; 1975.

The literature on the effect of various occupations on the occurrence of cancer of the larynx is reviewed. Interesting cases are cited that show the harmful effects which can result from exogenous or endogenous agents, or from overuse or abuse of the larynx. Exogenous agents, physical or chemical, affect the larynx either directly through breathing or indirectly through the skin. Of the physical agents, heat is considered by some to be the major factor, and it affects stokers, blast furnace workers, hot metal workers, engineers on steam locomotives, tar and asphalt workers, iron smelters, welders, blacksmiths, bakers, glass blowers, etc. Shortwave and UV radiation (700-14,000 μm) was found by others to have the greatest effect. The arsenic compounds, beryllium, chrome, and poisonous gases are the major chemical causes for cancer of the larynx. It is very difficult to find a cancer that can be attributed exclusively to an occupation. The literature offers no case of cancer of the larynx in professional singers or announcers. All occupations suspected being dangerous should be surveyed, taking tobacco and alcohol consumption into account.

4132 METASTATIC SITES THAT PRODUCE GENERALIZED CANCER: IDENTIFICATION AND KINETICS OF GENERALIZING SITES.

(Eng.) Bross, I. D. J.; Blumenson, L. E. *In: Fundamental Aspects of Metastasis.* Weiss, L., ed. (Amsterdam: North-Holland Publishing Co.): pp.359-375; 1976.

For most human cancers metastasis is a cascade process involving a sequence of two or more steps, in which the primary tumor first metastasizes to one or more generalizing sites that then secondarily seed the remainder of the body organs. Depending on the primary site, the generalizing sites may be the lung, liver, bones, spleen, or other organs. Cascade analysis on good autopsy data can reveal the generalizing sites and cascade sequence for a given primary. It is demonstrated that the lung is the generalizing site for head and neck cancers. An alternative method of deriving information about the kinetics of metastatic process from clinical, rather than autopsy, data is provided by use of the "deep mathematical model," which is outlined. (33 references)

4133 ANALYSIS OF THE EHRLICH ASCITES TUMOR GROWTH KINETICS.

(Ukr.) Ginzburg, Iu. E. (Inst. Problems Oncology, Acad. Sci. Ukranian SSR); Kaz'min, S. D. *Dopov Akad Nauk Ukr RSR Ser B* (1): 71-74; 1976.

A mathematical analysis of the growth kinetics of a tumor cell population under low glucose conditions is presented. A differential equation for deriving the cell number takes into account the cell reproduction rate in an aerobic subpopulation and glucose levels and utilization. The mathematical analysis was applied to Ehrlich ascites tumor cells in a constant proliferative pool and in a situation of decrease in population growth (from 100% to 4%). Glucose insufficiency was the major factor in decreasing ascites cell proliferation.

4134 POPPER'S PHILOSOPHY FOR EPIDEMIOLOGISTS (Letter to Editor). (Eng.) Buck, C. (Faculty Medicine, Univ. Western Ontario, London, Canada). *Int J Epidemiol* 5(1): 97-98; 1976.

4135 MORTALITY FROM LUNG CANCER AND CORONARY HEART-DISEASE IN RELATION TO CHANGES IN SMOKING HABITS. (Eng.) Wald, N. J. (Radcliffe Infirmary, Oxford OX2 6HE, England). *Lancet* 1(7951): 136-138; 1976.

4136 MORTALITY FROM LUNG CANCER. REPLY (Letter to Editor). (Eng.) Wald, N. (Radcliffe Infirmary, Oxford OX2 6HE, England). *Lancet* 1(7962): 742; 1976.

4137 COMBINED EFFECT OF FINE DUST ON THE LUNG. (Ger.) Einbrodt, H. J. (Abteilung Hygiene und Arbeitsmedizin, RWTH Aachen, Aachen, West Germany). *Staub Reinhaltung Luft* 36(3): 122-126; 1976.

4138 PULMONARY CYTOPATHOLOGY OF FORMER ASBESTOS WORKERS (Meeting Abstract). (Eng.) Greenberg, S. D. (Baylor Coll. Medicine, Houston, TX 77025); Hurst, G. A.; Matlage, W. T.; Christianson, C. S.; Hurst, I. J.; Mabry, L. C. *Am J Clin Pathol* 65(2): 257; 1976.

4139 SECONDARY RADIOGENIC CANCER OF THE LARYNX. (Ger.) Glanz, H. (Hals-Nasen-Ohrenklinik, Philips-Universitaet, Deutschausstrasse 3, D-3550 Marburg/Lahn, West Germany); Kleinsasser, O. *Z Hals Nasen Ohrenheilkd* 24(2): 48-59; 1976.

4140 CERVICAL CANCER AND EARLY SEXUAL INTERCOURSE (Letter to Editor). (Eng.) Peto, R. (Radcliffe Infirmary, Oxford, OX2 6HE, England). *Int J Epidemiol* 5(1): 97; 1976.

4141 AGE DISTRIBUTION OF PATIENTS WITH CARCINOMA OF THE COLON IN A GENERAL HOSPITAL IN IRAN. (Eng.) Vakili, C. (Pars Hosp., 147 Elizabeth II Blvd., Tehran, Iran); Fatourechi, V. *Surgery* 79(1): 118-119; 1976.

4142 CARCINOMA AFTER GASTRIC OPERATIONS. (Ger.) Clemencon, G. (CH-4600 Olten, Frohheimweg 12, Switzerland); Baumgartner, R.; Leuthold, E.; Miller, G.; Neiger, A. *Dtsch Med Wochenschr* 101(27): 1015-1020; 1976.

4143 FREQUENCY OF HOSPITALIZATION FOR TREATMENT OF CARCINOMA IN SITU IN EAST GERMANY. (Ger.) Blumenthal-Barby, K. (Akademie fuer Aerztliche Fortbildung der DDR, DDR-1134 Berlin, Noldnerstr. 34-36, East Germany); Rothe, J. *Zentralbl Gynaekol* 96(7): 398-402; 1974.

4144 THYROID AND OTHER AUTOANTIBODIES IN BRITISH AND JAPANESE WOMEN: AN EPIDEMIOLOGICAL STUDY OF BREAST CANCER. (Eng.) Mittra, I. (Guy's Hosp., London SE1 9RT, England); Perrin, J.; Kumaoka, S. *Br Med J* 1(6004): 257-259; 1976.

4145 QUANTITATIVE DETERMINATION OF SACCHARIN IN FOOD PRODUCTS BY ULTRAVIOLET SPECTROPHOTOMETRY. (Eng.) Hussein, M. M. (Life Savers, Inc., Port Chester, NY 10573); Jacin, H.; Rodriguez, F. B. *J Agric Food Chem* 24(1): 36-40; 1976.

See also:

* (Rev): 3601, 3605, 3606, 3607, 3608, 3609, 3610, 3611, 3612, 3613, 3614, 3616, 3617, 3618, 3619, 3620, 3621, 3622, 3623, 3630, 3637, 3644, 3649, 3659, 3660, 3670, 3673, 3676, 3678, 3684, 3686, 3689, 3691, 3693, 3697, 3698, 3699, 3700, 3721, 3724, 3731, 3732, 3733, 3734, 3735
* (Chem): 3746, 3759, 3760, 3761, 3768, 3785
* (Phys): 3834
* (Viral): 3917
* (Path): 4064, 4065, 4067, 4068, 4070, 4101, 4102, 4103

4146 CHARACTERISTICS OF 21S AND 7.5S RIBONUCLEOPROTEIN PARTICLES OF THE CYTOPLASM OF NORMAL AND TUMOR CELLS.

(Rus.) Roslyi, I. M. (Dept. Biochemistry, I. M. Sechenov First Moscow Medical Inst., Moscow, USSR). *Vopr Med Khim* 22(1): 113-116; 1976.

Cytoplasmic ribonucleoprotein (RNP) particles with 21S and 7.5S sedimentation values were isolated from tumors (sarcoma M-1, sarcoma 45, Jensen's sarcoma, and hepatoma 27), from normal rat brain, liver, and spleen tissue, and from a human tumor. All the RNP particles contained ribonucleoproteins similar in sedimentation characteristics to those from sarcoma M-1 cells. Ultracentrifugation of 21S and 7.5S particles of sarcoma M-1, sarcoma 45, and rat brain and liver in a 15%-30% sucrose density gradient showed that the particles in the 7.5S fraction were nonhomogeneous, in contrast to 21S particles. Polyacrylamide gel electrophoresis revealed the similarity of the protein components of 21S particles from various tissues; marked similarity was found also for the combined fractions of the proteins of 21S and 7.5S particles. Further confirmation of similarity was obtained by comparison of the incorporation of ^{14}C-leucine into RNP particles of normal (brain) and tumor (sarcoma M-1) cells 7 hr after introduction of the isotope. Incorporation took place significantly earlier in the 21S and 7.5S particle fractions than in the ribosomal fraction in normal as well as cancerous tissue.

4147 EFFECTS OF LOW TEMPERATURE ON RNA METABOLISM IN DIFFERENT CELL LINES.

(Eng.) Sensky, T. E. (Faculty Medical Sciences, Univ. Coll. London, London WC1E 6BT, United Kingdom); Haines, M. E.; Rees, K. R. *Exp Cell Res* 99(1): 200-204; 1976.

The behavior of RNA and the uridine nucleotide pools during and after a period of 4 C washing in six different monolayer cell lines was examined. Chinese hamster kidney (CHK), transformed CHK, BALB/c 3T3 mouse embryo strain XTY, transformed 3T3, HeLa, and African green monkey kidney (CV1) cell lines were given fresh medium without serum and containing ^{32}P, and incubated at 4 C for 3 hr. After return to 37 C, the RNA synthesis rate was measured during a 15-min pulse with ^{3}H-uridine. The cold treatment had no effect on the CHK cells, except for an apparent inhibition of ^{3}H-uridine incorporation into RNA, which gradually recovered. There was no label incorporation into acid-insoluble material at 4 C and no observable net exchange between the nucleotide pools and the medium. This was also true of the HeLa cells. The CVI and HeLa cells occasionally failed to divide after the treatment, and the transformed 3T3 cells were especially susceptible to lethal damage on rewarming. Of the effects studied, the only one common to many cell lines is that in cells prelabeled with ^{3}H-uridine, the total cellular acid-insoluble specific activity remained constant throughout the 4 C wash. This was true of the CHK cells and HeLa monolayers.

4148 CLONAL VARIATION IN ALBUMIN MESSENGER RNA ACTIVITY IN HEPATOMA CELLS.

(Eng.) Peterson, J. A. (Oakland Children's Hosp., 51st and Grove Sts., Oakland, CA 94609). *Proc Natl Acad Sci USA* 73(6): 2056-2060; 1976.

Clonal variation in the rate of albumin synthesis in hepatoma cells was used to investigate the epigenetic control of differentiation. In previous studies, variant subclones that produce albumin at different rates were readily isolated from a population of hepatoma cells. In the present experiments, in which a cell-free protein-synthesizing system from wheat germ was used, albumin messenger RNA activity was found to be directly proportional to the albumin synthesis rate in three different hepatoma clones, suggesting a pretranslational control of albumin production. Two hypotheses are given to explain the geometric pattern of clonal variation with respect to the organization and control of the transcriptional unit: (a) a ''regulatory'' gene is responsible for a regulatory sequence, which, if repetitive and metastable, could directly affect the transcription rate by determining the number of polymerase binding sites; and (b) there is a reflection of the molecular organization of chromatin that, if changed, would expose only part of a reiterated sequence for transcription. (43 references)

4149 EFFECT OF CELL PROLIFERATION ON LEVELS AND DIVERSITY OF POLY(A)-CONTAINING mRNA.

(Eng.) Getz, M. J. (Dept. Pathology, Mayo Clinic and Foundation, Rochester, MN 55901); Elder, P. K.; Benz, E. W., Jr.; Stephens, R. E.; Moses, H. L. *Cell* 7(2): 255-265; 1976.

The relationship between cell proliferation and the amount and diversity of polyribosome-associated poly(A)-containing messenger RNA [poly(A)+mRNA] was investigated using a cloned AKR mouse embryo cell culture. The following results were obtained. First, an early response to the stimulation of proliferation of AKR-2B cells in culture was a rapid increase in the rate of accumulation of polyribosomal poly(A) + mRNA. This results in a large increase in the total poly(A)+ mRNA content of rapidly proliferating cells compared to that found in resting cells. Second, the total amount of unique DNA sequence contributing to the poly(A)+ mRNA populations of both growing and resting cells was not detectably different. This corresponded to 9000-11,000 diverse gene equivalents of DNA and represented the transcription of 0.8-0.9% of the haploid mouse genome. Third, most of the increased poly(A)+ mRNA content of growing cells (> 90%) reflected an increased rate of production of polysomal mRNA species which are also found in resting cells. Fourth, growing cells appeared to contain some species of poly(A)+ mRNA which were either absent or present in very low concentrations in nongrowing cells. Within the limits of detection, however, all species of poly(A)+ mRNA present in nongrowing cells were also present in growing cells.

4150 CHANGES IN THE DEOXYADENYLATE REGIONS IN 5 CASES OF CHRONIC LYMPHOCYTIC LEUKEMIC DNA.

(Eng.) Pero, R. W. (Nucleic Acids Res. Group, Univ.

Lund, Box 740, 220 07 Lund, Sweden); Bryngelsson, T.; Deutsch, A.; Norden, A. *J Cancer* 12(5): 357-362; 1976.

The deoxyadenylate (dA) levels in the DNAs from normal human tissues and from the lymphocytes of five chronic lymphocytic leukemia (CLL) patients were estimated by the formation of ribonuclease A-resistant polyuridylic acid (poly U)-DNA complexes. Alterations in the dA levels of all five CLL DNAs were observed when comparison was made to the normal dA pattern. The type of dA alterations varied among the different CLL patients, but the variation correlated to the WBC levels of the patients. A specificity of neoplastic-associated dA alterations for slow repeated and unique DNA sequences and for main band DNA was demonstrated with two of the CLL DNAs. Since most mRNA is transcribed from slow repeated or unique DNA sequences, the dA changes observed in these DNA fractions may reflect the differences in RNA transcription that have previously been described between normal and CLL lymphocytes. (23 references)

4151 SINGLE-STRANDED REGIONS IN REGENERATING RAT LIVER DNA. (Eng.) Hoffman, L. M. (Medical Coll. Virginia, Virginia Commonwealth Univ., Richmond, VA 23298); Collins, J. M. *Nature* 260(5552): 642-643; 1976.

Isopycnic cesium sulfate gradients containing silver ion and a single-strand-specific endonuclease were used to determine the single strandedness of DNA from actively regenerating rat liver and from the liver of partially hepatectomized rats that had been allowed to regenerate over 95% of their normal liver mass (control liver). Male Sprague-Dawley rats 1 mo old were subjected to partial hepatectomy; the actively regenerating liver was obtained from seven animals killed 20.5 hr later, and regenerated liver was obtained from seven animals killed 14 days later. DNA from actively proliferating liver seemed to be more single-stranded than DNA from control liver, because the 20.5-hr liver had a greater buoyant density than that of 14-day regenerating DNA by 0.09 g cm^{-3}. DNA obtained 20.5 hr after hepatectomy contained three times more single-strand nuclease-sensitive sites than DNA from 14-day regenerate livers. The physiological significance of single-stranded regions in regenerating DNA is unclear, but they are likely to be involved in DNA replication.

4152 NUCLEAR DNA-POLYMERASE ESTIMATION IN HUMAN LEUKAEMIC MYELOBLASTS. (Eng.) Wantzin, G. L. (Rigshospitalet 9, Blegdamsvej, DK-2100 Copenhagen, Denmark); Karle, H.; Killmann, S.-A. *Br J Haematol* 33(3): 329-334; 1976.

The presence of DNA-polymerase in cell nuclei was measured by the incorporation of ^{3}H-thymidine-5-triphosphate (^{3}H-TTP) in order to estimate the proportion of leukemic myeloblasts containing DNA-polymerase and DNA capable of acting as primer-template. In six cases of previously untreated acute myeloid leukemia, the ^{3}H-TTP labeling index (^{3}H-TTP LI) was much larger than the fraction in DNA synthesis. After a single flash injection of cytosine arabinoside, a pronounced decline was observed in the ^{3}H-TTP LI, which can be explained by a direct inhibition of DNA-polymerase. No change was observed in the fraction of cells labeled with ^{3}H-thymidine. A decrease in ^{3}H-TTP LI was also observed after a single iv dose of methotrexate. It is believed that ^{3}H-TTP single cell assay can characterize the effect of cytostatics on cell growth potential. (12 references)

4153 MICROSOMAL FUNCTIONS AND PHENOTYPIC CHANGE IN ADULT RAT HEPATOCYTES IN PRIMARY MONOLAYER CULTURE. (Eng.) Bissell, D. M.; Guzelian, P. S. *In: Gene Expression and Carcinogenesis in Cultured Liver.* Gerschenson, L. E.; Thompson, E. B., eds. (New York: Academic Press, Inc): pp. 119-136; 1975.

A method for preparing adult rat hepatocytes in primary monolayer culture is reviewed and the specific characteristics of the cells are described. NADPH-cytochrome C reductase, cytochrome P-450, aminopyrine N-demethylase, benzo(a)pyrene hydroxylase, aniline hydroxylase, and heme oxygenase were measured. When the cell layers were examined for mitotic figures at intervals during the first 48 hr of incubation (at 35 C under air), only one mitosis in approximately 10^5 cells was seen; the high rate of DNA synthesis at the time of plating may be an artifact. Several functions remain near in vivo levels in the culture system for several days: albumin synthesis and secretion; glucogenesis from 3-carbon precursors; glycogen synthesis; responsiveness to insulin and glucagon; p-nitroanisole O-demethylase induction; bile salt conjugation; uptake and storage of sulfobromophthalein; and heme catabolism to bilirubin. Over the first 24 hr the cellular concentration of cytochrome P-450 decreased to approximately 20% of its initial level; this apparently did not indicate generalized metabolic deterioration of the cultured cells because of the preservation of numerous functions and overall protein synthesis. It is suggested that although hormones or drugs may modulate the regulation of cytochrome P-450 in vivo, the regulatory mechanism itself is primarily subject to the internal metabolic structure of the hepatocytes. This study has implications for hepatic physiology in vivo; for example, the apparently reciprocal relationship between the concentration of cytochrome P-450 and the activity of heme oxygenase in the liver was not previously appreciated. (28 references)

4154 L-LEUCINE, A SPECIFIC INHIBITOR OF A RARE HUMAN PLACENTAL ALKALINE PHOSPHATASE PHENOTYPE. (Eng.) Doellgast, G. J. (Tufts Univ. Sch. Medicine, 136 Harrison Ave., Boston, Mass. 02111); Fishman, W. H. *Nature* 259(5538): 49-51; 1976.

Placentas were screened for the presence of a rare serum D-phenotype, L-leucine-sensitive alkaline phosphatase. A sample of ovarian cancer ascitic fluid containing the variant was used as a reference standard. Nine placentas of 2,323 screened contained an enzyme that closely resembled the cancer patient enzyme in sensitivity to L-leucine. The 2,314 other placentas were not inhibited by L-leucine, while the variant enzymes reached a maximum of 40% inhibition at 0.5-0.2 mM L-leucine. Although the leucine sensitivities of the cancer patient enzyme and the placental variant appear similar, this alone is not proof that the two enzymes represent the same genomic origin or that the enzyme reflects a predisposition to certain cancers.

4155 ACID PHOSPHATASE OF HELA CELLS: PROPERTIES AND REGULATION OF LYSOSOMAL ACTIVITY BY SERUM.

(Eng.) Wang, C.-C. (Dept. Molecular Biology, Vanderbilt Univ., Nashville, TN 37235); Touster, O. *Arch Biochem Biophys* 172(1): 191-201; 1976.

Although the subcellular distribution profile of acid phosphatase in HeLa cells is typical of a lysosomal enzyme, different lysosomal (70-80%) and supernatant forms (20-30%) were demonstrated by their differences in pH activity curves, substrate specificities, thermal stability, sensitivity to inhibitors, and kinetics. Enzymes of the lysosomal fraction displayed anomalous kinetics in the hydrolysis of p-nitrophenyl phosphate. The major lysosomal acid phosphatase activity appears to be associated with the membrane. The total acid phosphatase activity in the cell is controlled by the concentration of serum in the medium. The specific activity in the homogenates of cells grown in high serum concentration (30%) was about twice that of cells grown in low serum concentration (1%). This doubling of specific activity held for the lysosomal enzyme (or enzymes), but little change occurred in the supernatant form (or forms). Two other lysosomal enzymes, beta-glucuronidase and N-acetyl-beta-D-hexosaminidase, do not increase in specific activity. The serum-dependent formation of acid phosphatase was sensitive to cycloheximide, actinomycin D, and cordycepin. Cycloheximide blocked the increase in enzymatic activity immediately, whereas cordycepin and actinomycin D had no effect for at least 8 hr. These findings suggest that de novo protein synthesis is involved in the induction of lysosomal acid phosphatase by serum and that the mRNA for this enzyme is relatively stable.

4156 IDENTIFICATION OF HEXOKINASE SYNTHESIZING POLYRIBOSOMES IN HUMAN NORMAL AND TUMOR TISSUES.

(Rus.) Monakhov, N. K. (Inst. Experimental Medicine, Acad. Medical Sciences USSR, Leningrad, USSR); Shvartsman, A. L.; Mukha, G. V.; Kiselev, O. I.; Gaitskhoki, V. S.; Neifakh, S. A. *Biokhimiia* 41(4): 589-596; 1976.

The concentrations of growing polypeptide chains of hexokinase subunits were studied in polyribosomes from normal and tumor stomach tissue with the use of a specific antiserum to hexokinase isoenzyme from the tumor tissue. Chromatography on Sepharose 46 and sucrose concentration gradient analysis were employed. Identical amounts of polyribosomes from normal and tumor tissue bound different amounts of antihexokinase immunoglobulins. The content of hexokinase polypeptides was several times greater in the tumor tissue than in homologous normal tissue because of the increased rate of synthesis of this enzyme. The increased amount of hexokinase-synthesizing polyribosomes in the cancer cells may reflect the heightened concentration of growing hexokinase chains reacting with antibodies. The data also suggest an increase in the translation effectiveness of each hexokinase-messenger RNA molecule in tumor tissue, accompanied by an increase in the size of corresponding polyribosomes to a theoretical threshold value.

4157 TWO FORMS OF THYMIDINE KINASE IN NORMAL AND TUMOR TISSUES OF ANIMALS.

(Eng.) Ohashi, M. (Tokyo Metropolitan Inst. Gerontology, 35-2 Sakae-cho, Itabashi-ku, Tokyo 173, Japan); Taguchi, T. *Cancer Res* 36(7): 2216-2222; 1976.

The thymidine kinases extracted from the spleen of C3H/He mice infected with Friend virus and from Yoshida sarcoma in Wistar rats were separated into two active peaks by diethylaminoethylcellulose column chromatography, while those of normal tissues were found to consist of only the first peak (P I). The second peak (P II) was also found in the enzyme from a spleen extract when the animals were treated by ip injections of 1-acetyl-2-phenylhydrazine, which causes severe hemolytic anemia and splenomegaly in mice and rats. The two P II peaks from tumor tissue and from the spleen enlarged by anemia-inducing agents were indistinguishable on the chromatographic profile. On the other hand, the thymidine kinase extracted with Triton X-100 from a mitochondrial fraction of normal liver consisted of only one peak in the same position as the above P II's , but its faculty for deoxythymidine triphosphate inhibition was not identical to that of the tumor tissue. Treatment with the detergent might dissociate a certain component from the enzyme complex to make the extra peak (P IIb), but it eventually shifts to P II on the chromatogram. (40 references)

4158 β-GLUCURONIDASE ISOZYME PATTERNS OF EXPERIMENTAL HEPATOMAS OF RATS.

(Eng.) Kakizoe, T. (Faculty Medicine, Univ. Tokyo, Hongo 7-3-1, Bunkyo-ku, Tokyo 113 Japan); Kawachi, T.; Sugimura, T. *Gann* 67(2): 289-294; 1976.

β-Glucuronidase isozymes in Buffalo strain rat tissues were separated by electrophoresis on cellulose acetate membranes, and their patterns were studied in normal rat liver and experimental hepatomas with different growth rates. Changes of isozyme patterns during postnatal development were also studied in the rat liver. Normal rat liver contained six types of β-glucuronidase, numbered I to VI in order of decreasing mobility to the cathode. Type II β-glucuronidase stained most intensely and was detected in all the cells examined. The isozyme patterns of Morris hepatomas, which are slowly growing and not highly deviated, resembled those of normal rat liver but lacked definite type III β-glucuronidase. All Yoshida ascites hepatomas, which are rapidly growing and highly deviated, contained type II, but some also had types II, V, and VI β-glucuronidase. It is concluded that there is a correlation between β-glucuronidase enzyme patterns and tumor growth rates. Embryonal liver just before and up to 6 days after birth contained only type II, but with increase in activity during development after birth, minor bands of type I and types III to VI β-glucuronidase appeared successively to complete the adult pattern at 65 days. (33 references)

4159 DIFFERENCES BETWEEN PYRIMIDINE NUCLEOSIDE MONOPHOSPHATE KINASE FROM RAT NOVIKOFF ASCITES HEPATOMA AND RAT LIVER.

(Eng.) Maness, P. F. (Univ. Texas System Cancer Center, M. D. Anderson Hosp. Tumor Inst., Houston, TX 77030); Orengo, A. *Cancer Res* 36(7): 2312-2316; 1976.

Pyrimidine nucleoside monophosphate kinase [deoxycytidine monophosphate:adenosine triphosphate (dCMP: ATP) phosphotransferase] has been purified from rat Novikoff ascites hepatoma and rat liver from female Holtzman Sprague-Dawley rats. Purity of the hematoma and rat liver enzyme fraction was measured by comparison to a single major band appearing on sodium dodecyl sulfate-polyacrylamide gel electrophoresis. Differences exist in regard to efficiency and regulation of enzymatic activities. The Km values of the tumor kinase for cytidine monophosphate (CMP) and dCMP are approximately one-fourth the Km values of the rat liver kinase and dCMP. The tumor dCMP kinase exhibits a lower Km for ATP than the rat liver kinase. Moreover, the dCMP:CMP kinase activity ratio for the tumor enzyme is 1.12, while that for the rat liver enzyme is 0.45. The uridine monophosphate:CMP kinase activity ratio for the tumor enzyme is 1.93, while that for the rat liver enzyme is 2.68. Lower concentrations of dithiothreitol are required for 50% reactivation of the tumor dCMP kinase (1.00 mM) and CMP kinase (0.10 mM) than rat liver dCMP kinase (2.20 mM) and CMP kinase (0.57 mM). Thus, the kinase from Novikoff hepatoma exhibits properties of increased efficiency and relaxed regulation of activity that render it more suitable for a tumor in which active DNA synthesis is ongoing. (16 references)

4160 STIMULATORY EFFECT OF 3'5'-ADENOSINE MONOPHOSPHATE ON PROTEIN SYNTHESIS IN HEAT-SHOCKED MURINE LEUKEMIA LYMPHOBLASTS.

(Eng.) Fuhr, J. E. (Memorial Res. Center, Univ. Tennessee, 1924 Alcoa Highway, Knoxville, TN 37920); Overton, M.; Yang, T. J. *J Natl Cancer Inst* 56(1): 189-191; 1976.

The murine leukemia lymphoblast line L5178Y was tested for its early molecular response to the presence of dibutyryl cyclic AMP. Protein synthesis was reversibly inhibited by short durations of hyperthermia (44 C), and against this reduced background of protein synthetic activity, early leucine incorporation (30-120 min) was markedly and consistently stimulated by the presence of the cyclic nucleotide. The effect became one of inhibition with extended times of incubation (longer than 120 min).

4161 CHOLESTERYL ESTER METABOLISM IN TISSUE CULTURE CELLS: II. SOURCE OF ACCUMULATED ESTERIFIED CHOLESTEROL IN FU5AH RAT HEPATOMA CELLS.

(Eng.) Rothblat, G. H. (The Wistar Inst., 36th and Spruce Sts., Phila., PA 19104); Arbogast, L.; Kritchevsky, D.; Naftulin, M. *Lipids* 11(2): 97-108; 1976.

The origin of cellular esterified cholesterol in Fu5AH rat hepatoma cells was established, and the interaction of the cells with hyperlipemic serum proteins was examined. In normal rabbit serum (NRS) the cellular free cholesterol content of the hepatoma cells is not significantly altered, whereas it is increased twofold in hyperlipemic rabbit serum (HRS). Hepatoma cells in hyperlipemic rabbit serum derive esterified cholesterol from three sources: (a) 10% from endogenous free cholesterol, (b) 30% from exogenous free cholesterol, and (c) 60% from exogenous cholesterol esters. Modified very low density or intermediate lipoproteins isolated from hyperlipemic rabbit serum induce the greatest esterified cholesterol influx and the greatest free cholesterol influx and esterification. Tween 80 and Triton WR-1339 reduce the effect of the hyperlipemic serum. The accumulation of esterified cholesterol, the incorporation of free cholesterol, and the esterification of cellular cholesterol were compared in rabbit aortic medial cells, L-cells, and Fu5-5 rat hepatoma cells. In NRS the L, aortic medial, and Fu5-5 cells showed <1% esterification in contrast to 31% in Fu5AH cells, which also had somewhat higher esterified cholesterol levels. HRS stimulated esterification and increased free and esterified cholesterol levels in all cells, with Fu5AH showing the highest cellular esterified cholesterol level and the greatest percent of esterification.

4162 ANIMAL MODELS OF ENDOCRINE-RELATED CANCER.

(Eng.) Pearson, O. H. *In: Host Environment Interactions in the Etiology of Cancer in Man. Proceedings of a Meeting Held at Primosten, Yugoslavia, 27 August-2 September 1972.* International Agency for Res. on Cancer (Lyon, France): pp. 137-141; 1973.

The use of two rat mammary tumors to evaluate the endocrine factors involved in the induction and growth of rat mammary cancer, which may provide a guide to the endocrine factors involved in human breast cancer, is reviewed. One tumor, induced by 7,12-dimethylbenz(a)anthracene (DMBA), is prolactin-dependent. The similarities between this cancer and hormone-responsive breast cancer in women suggest that the same endocrine factor may be operative; however, preliminary studies suggest that the regulation of prolactin secretion differs in humans. For example, the preovulatory rise in prolactin secretion in the rat estrus cycle has not been noted in the human menstrual cycle. The growth of the R3230AC rat mammary tumor is slowed by the administration of ovine prolactin. It appears that prolactin may have a dual effect on tumor growth, at least in animal models. (17 references)

4163 ANDROGEN-DEPENDENT FIBRINOLYTIC ACTIVITY IN A MURINE MAMMARY CARCINOMA (SHIONOGI SC-115 CELLS) IN VITRO.

(Eng.) Mak, T. W. (Dept.

Medical Biophysics and Medicine, Univ. Toronto, Toronto, Ontario M4X 1K9, Canada); Rutledge, G.; Sutherland, D. J. *Cell* 7(2): 223-226; 1976.

Physiological concentrations of dihydrotestosterone (DHT) could specifically induce the release of fibrinolysin from androgen-dependent mammary carcinoma (Shionogi SC-115) cells in vitro. No fibrinolytic activity was observed in cells cultured either in the absence of DHT or presence of pharmacological concentrations of estrogen. Furthermore, an autonomous tumor derived from the Shionogi SC-115 cells produced fibrinolytic activity independent of added DHT or estrogen. These observations suggest a close correlation between fibrinolytic activity of a tumor and its ability to grow in vivo.

4164 GROWTH HORMONE AND DRUG METABOLISM. (Eng.) Wilson, J. T. (Dept. Pediatrics, Vanderbilt Sch. Medicine, Nashville, TN 37232); Spelsberg, T. C. *Biochem J* 154(2): 433-438; 1976.

Adult male rats were subjected either to sham operation or to hypophysectomy and adrenalectomy and maintained for a total of 10 days before treatment with growth hormone. Hypophysectomy and adrenalectomy resulted in decreased body and liver weight and decreased drug metabolism (mixed-function oxidases). Concentrations of electron-transport-system components were also decreased. In the hypophysectomized/adrenalectomized rats, growth hormone decreased the activities of the liver mixed-function oxidases and the cytochrome P-450 and cytochrome c reductases, as well as decreasing the concentration of cytochrome P-450 compared with that of control rats. Similar but less dramatic results were obtained with sham-operated rats. It is concluded that whereas growth hormone enhances liver growth, including induction of many enzyme activities, it results in a decrease in mixed function oxidase activity. Apparently, mixed function oxidase activity decreases in liver when growth (mitogenesis) increases.

4165 REGULATION OF ENZYME TURNOVER DURING TISSUE DIFFERENTIATION. (Eng.) Speake, B. K. (Dept. Biochemistry, Univ. Hosp. and Medical Sch., Clifton Boulevard, Nottingham NG7 2UH, U.K.); Dils, R.; Mayer, R. J. *Biochem J* 154(2): 359-370; 1976.

Explants of mammary gland from mid-pregnant rabbits were cultured with insulin, prolactin and cortisol. With hormone combinations which included prolactin, a sustained increase in the apparent rate of synthesis and in the amount of fatty acid synthetase was measurable immunologically. Maximum increase was produced with insulin, prolactin and cortisol present together. With prolactin present alone, synthetase activity in the explants decreased to undetectable values after 1 day in culture, whereas the incorporation of L-[U-^{14}C]leucine into immunodetectable material increased. Prolactin may therefore direct the synthesis of immunologically cross-reactive precursors of fatty acid synthetase which are enzymatically inactive. Culture with dibutyryl cyclic AMP plus theophylline in the presence of insulin, prolactin and cortisol delayed the increase in the rate of synthesis and accumulation of the synthetase. These compounds may also prevent the apparent decrease in the rate of degradation of the synthetase which occurs on day two of culture. This decrease in the apparent rate of degradation of the synthetase was measured by radioimmunological precipitation. It is probably part of a regulated program of enzyme degradation and not a reflexion of the reutilization of radioactive amino acids. It is concluded that there is a real decrease (or even cessation) in the rate of degradation of fatty acid synthetase during the period when the enzyme accumulates in explants cultured with hormone combinations which contain prolactin.

4166 EFFECT OF AMINO ACIDS, INSULIN, AND GLUCAGON ON THE GROWTH OF CELLS IN VITRO. (Eng.) Demetriou, A. A. (Dept. Surgery, Albert Einstein Coll. Medicine, Yeshiva Univ., Bronx, NY); Thyssen, B.; Schittek, A.; Seifter, E.; Rettura, G.; Levenson, S. M. *Surg Forum* 26: 184-186; 1975.

The effect of various solutions of amino acids, glucagon, and insulin on the in vitro growth of E_1 cells derived from fetal rat liver was studied. The E_1 cells were grown at 37 C in 5% CO_2, and the initial cell inoculum was 20,000 cells. Cell growth was stimulated significantly when a solution of amino acids in concentrations found in the sera of 70% hepatectomized rats was added to the culture medium. Glucagon (70 μg/100 ml) and/or insulin (1.6 μg/100 ml) also stimulated the E_1 cell growth in vitro, and their effects were additive to that of the amino acid mixture. A significant increase in systemic blood serum insulin (51.6 μU/ml) was observed 24 hr after 70% hepatectomy on Long Evans rats killed by withdrawal of heart blood. The serum insulin level of rats that underwent 99.5% pancreatectomy was significantly lower (11.0 μU/ml) than that of controls (22.2 μU/ml) or rats undergoing sham hepatectomy (midline abdominal incision and gentle palpation of the liver). Serum insulin values remained low (12.1 μU/ml) in rats that underwent 99.5% pancreatectomy followed by 70% hepatectomy 5 days later. (3 references)

4167 MEASUREMENT OF CELL GROWTH BY A RAPID U.V. SPECTROPHOTOMETRIC METHOD. (Eng.) Page, M. (Service de la Recherche, Hotel-Dieu de Quebec, 11, Cote du Palais, Quebec, Canada); Dufour, C. *Biomedicine [Express]* 25(5): 170-173; 1976.

A spectrophotometric method was used to measure mammalian cell growth in culture. Human epithelioid carcinoma cells (HeLa), normal human liver cells, human epidermoid carcinoma cells (KB), and rat hepatoma cells were used. The attached cells were solubilized in 2N sodium hydroxide, and the absorption was measured at 225 nm. The technique is sensitive to about 3,000 cells per petri dish, and a linear relationship was obtained between the number of cells and the absorption at this wavelength. The method is rapid, and the solubilized material can be recovered for liquid scintillation counting when needed. (10 references)

4168 THE ROLE OF BIOCHEMICAL RHYTHMS IN CONTACT INHIBITION OF CELLULAR DIVISION. (Eng.) Burton, A. C. *In: Cellular Membranes and Tumor Cell Behavior. Annual Symposium on Fundamental Cancer Research, 1975.* (Baltimore: Williams and Wilkins Co.): pp. 249-266; 1975.

A theory is presented to explain how intercellular communication could mediate contact inhibition of normal cells in tissue. The theory postulates that there is a key substance that has to reach threshold concentration for a cell to divide. It is assumed that the key substance moves from cell to cell by simple diffusion, that the cell has normal or suppressed intrinsic oscillation, and that the intrinsic oscillations of individual cells are asynchronous. This theory of diffusion-coupled asynchronous biochemical oscillations suggests that there could be three basic causes of cancer: excessive intrinsic oscillations, synchrony of groups of cells, or lack of normal communication. Analysis of a simple mathematical model of two cells in contact with an infinite sheet of communicating cells led to the predictions that in small aggregates of cells, contact facilitation may exist and favor survival and that synchrony of cells, as in embryonic tissue, may explain lack of contact inhibition where there is normal communication. A coefficient of variation of ± 5% in the intrinsic periods of the cells is sufficient for marked inhibition (in normal cells the variation is higher). It is suggested that the key substance must meet the following requirements: (a) be a relatively small molecule; (b) affect a great number of enzymic reactions that control synthesis within the cells; and (c) fluctuate periodically in cellular concentration with the period of the cell cycle. Acid-base relations in the cell satisfy the first two requirements, and recent studies indicating a sinusoidal swing of pH in the cycle of a synchronized slime mold of more than 0.5 pH units suggest that the third requirement may be met as well. Acclimatization to high altitudes involves chronic changes in acid-base relations, and statistical data were examined that showed a strong, highly significant decrease in cancer with higher altitude. Other work on the effects of external pH on tissue culture growth when coupled with the altitude results suggests that manipulation of acid-base relations in cancer patients might lead to some control of neoplastic growth. (20 references)

4169 ENHANCEMENT OF PHENOTYPIC EXPRESSION IN CULTURED MALIGNANT LIVER EPITHELIAL CELLS BY A COMPLEX MEDIUM. (Eng.) Gunn, J. M. (Temple Univ. Medical Sch., Philadelphia, PA 19140); Shinozuka, H.; Williams*, G. M. *J Cell Physiol* 87(1): 79-87; 1976.

The ability of two media identically supplemented with 10% fetal bovine serum to maintain functional and structural differentiated properties of cultured malignant liver epithelial cells was compared. Reuber H35 hepatoma cells grown in a complex medium, Medium E, possessed the gluconeogenic enzyme phosphoenolpyruvate carboxykinase and bile canaliculi. When grown in Eagle's Minimal Essential Medium, the activity of phosphoenolpyruvate carboxykinase was reduced, and bile canaliculi were not formed. This decrease in properties was restored when cells maintained in Minimal Essential Medium were returned to Medium E. It is concluded that the enhanced phenotypic expression was probably due to enrichment of the nutrient environment.

4170 INTERCELLULAR COMMUNICATION IN NORMAL AND NEOPLASTIC TISSUES. (Eng.) Loewenstein, W. R. *In: Cellular Membranes and Tumor Cell Behavior. Annual Symposium on Fundamental Cancer Research, 1975.* (Baltimore: Williams and Wilkins Co.): pp. 239-248; 1975.

Studies investigating the relationship between junctional defects in cancerous tissues and growth defects are reviewed. It is postulated that control of growth involves four elements: control signals, signal transmission, signal receptors, and effector processes. Junctional defects in signal transmission were identified in six cancerous cell strains, four derived from rat hepatomas, one produced by x-irradiation of hamster embryo cells, and one a malignant derivative of a mouse L cell. All six strains are also abnormal in their growth patterns; they do not show density-dependent growth in culture and they produce fatal tumors in animals. Cells from any of these strains will not couple with one another or with cells from different organs and species. Hybridization studies using cells from the junction defective strains fused with normal cells indicated that density-dependent growth is resumed and tumorigenicity reduced in the hybrids (human skin fibroblast and the malignant derivative of a mouse L cell). The hybrids resembled the human parent, while they contained the nearly complete parent chromosome complement. However, the hybrid cells lost human chromosomes upon continued cultivation and clones eventually appeared that had reverted to the growth defective trait of the mouse parent; these clones had also reverted to the junction-defective trait. The normal human cell may contribute a genetic factor that corrects the growth and junction defects simultaneously. The junction defect and the in vitro growth defect are correlated in this cell system. (20 references)

4171 CHARACTERISTICS OF PRIMARY AND SERIALLY TRANSPLANTED TUMOURS AND DERIVED CULTURES. (Eng.) Deys, B. F. (Laboratorium voor Radiobiologie van de Universiteit van'Amsterdam, Amsterdam, The Netherlands); Barendsen, G. W. *Adv Tumour Prev Detect Charact* 3: 374-382; 1976.

Changes in cellular characteristics, both in vivo and in vitro, were investigated with the use of a keratinizing ureter carcinoma (RUC) and a mouse osteosarcoma (MOS). Cultured RUC cells progressed from fibroblasts to epitheloid-type cells that produced keratin granules. After 4-5 mo these epithelial cells became smaller, produced less keratin, and showed a degree of density-dependent inhibition. MOS cells lacked contact inhibition throughout the culture period, and various morphologically distinct clones of cells were present. Bone formation was not detected in the primary or passage cultures. The cloning efficiency was higher in MOS cells compared to RUC cells; however, MOS cells were more sensitive to irradiation. Both types of cells caused tumors when they were injected into BN/Bi rats and BALB/c mice, but the characteristics of both transplanted tumors were different from those of the original tumors. These changes may be due to the effects of the trypsin treatment used in cell transfer studies. It is concluded that research on transplantable tumors or long-term cell cultures derived from tumors may not always provide information directly relevant to treatment. (15 references)

4172 INVESTIGATION OF THE ACOUSTIC PARAMETERS OF BLOOD DURING THE DEVELOPMENT OF EXPERIMENTALLY INDUCED NEOPLASMS. (Rus.) Braginskaia, F. I. (Inst. Chemical Physics, Acad. Sciences USSR, Moscow, USSR); Minenkova, E. A.; Krugliakova, K. E. *Dokl Akad Nauk SSSR* 228(4): 966-969; 1976.

Ultrasonic absorptivity was measured in blood samples from the femoral vein of rats with experimental sc Walker carcinosarcoma. Acoustic parameters were also determined in the plasma after the cellular elements were removed by centrifugation. Absorptivity in the whole blood was gradually decreased during tumor development. Tumor growth did not affect the ultrasonic absorptivity in the blood plasma. It is concluded that the observed decrease was due mainly to absorption in the RBC. Growth of the Walker carcinosarcoma was correlated with the decrease in the number of RBC in blood, while an inverse correlation was observed for the WBC. (7 references)

4173 A MOLECULAR ORBITAL STUDY ON THE INTERACTION BETWEEN THE CYTOCHROME P-450 AND ITS SUBSTRATES. (Eng.) Nagata, C. (Natl. Cancer Center Res. Inst., Tsukiji, Chuoku, Tokyo 104, Japan); Fujita, H.; Imamura, A. *Chem Biol Interact* 12(1): 1-12; 1976.

Models for the interaction of the cytochrome P-450 with its substrates, namely for the type I interaction are proposed in which the molecular planes of the aromatic substrate and the prophyrins of P-450 are in parallel. Optical values such as transition energies and oscillator strengths for the isolated P-450 and P-450-substrate complex were calculated by means of molecular orbital method (ASMO SCF CI method), and they were compared with the observed values. The fact that no appreciable shift in the absorption peak of Soret band of P-450 was observed upon addition of substrate was reflected in the calculation. Similarly, the fact that the absorbance of the P-450 was decreased by mixing it with the substrate could also be explained by the calculated oscillator strength for the isolated P-450 and the stacked complexes. The agreement between the calculated and observed results suggests that the proposed models might be true reflections of the interactions between the P-450 and its substrates.

4174 LENS CULINARIS LECTIN IMMOBILIZED ON SEPHAROSE: BINDING AND SUGAR-SPECIFIC RELEASE OF INTACT TISSUE CULTURE CELLS. (Eng.) Kinzel, V. (Inst. Experimental Pathology, Deutsches Krebsforschungszentrum, Heidelberg, West Germany); Kuebler, D.; Richards, J.; Stoehr, M. *Science* 192 (4238): 487-489; 1976.

The ability of immobilized lens culinaris lectin to induce cultured HeLa and SV 3T3 cells to bind large agarose beads and to release them specifically on treatment with hapten sugars was investigated. Beads activated with cyanogen bromide and untreated beads failed to bind the cells. In contrast, beads treated with lens culinaris lectin were covered with bound cells within 10-30 min at room temperature. The hapten sugar methyl-α-D-glucopyranoside at 0.2 M inhibited the binding of the cells to the beads. The same concentration of methyl-α-D-mannopyranoside inhibited binding more strongly, preventing almost any binding. D-galactose had no effect. Cells bound to lectin-treated beads for 1 hr were specifically dislodged by methyl-α-D-glucopyranoside and methyl-α-D-mannopyranoside, more efficiently by the latter. A trypan blue exclusion test for cell integrity showed that a 2-hr binding and release procedure increased the percentage of trypan blue-positive cells by about 5% over control values. Released cells also multiplied in complete medium 1.54-fold in 24 hr, while control cells multiplied 1.65-fold. It is concluded that immobilized lens culinaris lectin induces cultured cells to bind to a solid matrix, but this is prevented by hapten sugars specific for the lectin.

4175 STUDIES ON THE METABOLISM OF CYTOPLASMAL BASIC PROTEINS WITH HIGH ARGININE LEVEL FROM GUERIN'S EPITHELIOMA. (Pol.) Farbiszewski, R. (Zaklad Biochemii Instytutu Fizjologii i Biochemii AM, 15-230 Bialystok 8, ul. Mickiewicza 2, Poland). *Pol Tyg Lek* 31(14): 595-596; 1976.

Basic protein rich in arginine was isolated from male Wistar rats with Guerin's epithelioma, and its physicochemical properties and degradation products were identified. The use of ^{14}C-1-arginine to study the metabolism indicated that peptide fragments containing arginine and appearing in the blood of rats with this neoplasm may result from degradation of cellular proteins in the tumor. The quantity of arginine-rich basic proteins was proportional to the stage of tumor advancement. In vitro studies showed that basic proteins were degraded most rapidly by proteases of lysosomal tumor extracts. Comparison of the results of autodegradation of labeled subcellular protein fractions in Guerin's epithelioma indicated that proteins were digested the fastest by cellular enzymes of the tumor. In vivo studies confirmed that the rate of incorporation of ^{14}C-arginine into proteins of Guerin's epithelioma cells was 25% greater than into proteins of the rat endometrium in rats receiving arginine 10, 20, or 30 days after inoculation with neoplastic cells. The findings indicate accelerated metabolism of basic proteins with high arginine levels in Guerin's epithelioma cells.

4176 LACTATE DEHYDROGENASE ACTIVITY IN SPLEEN CELL CULTURES OF NORMAL AND LEUKEMIC COWS. (Rus.) Kalnina, E. A. (Avgust Kirkenshtein Inst. Microbiology, Acad. Sciences Latvian SSR, USSR); Zhilevich, A. V. *Veterinariia* (3): 55-57; 1976.

The isozyme spectrum and general activity of lactate dehydrogenase (LDH) were studied in spleen cell cultures obtained from healthy and leukemic cows and in cell cultures of tissues of a cow embryo inoculated with an extract from the spleen of leukemic cows. The increase in the number of cell culture passages was accompanied by a change in the relationship of LDH isozymes in the cells. The percentage of cathode fractions slow in electrophoresis rose and the general activity of the enzyme increased. The increase in the percentage of the cathode fractions and the simultaneous decrease in anode fractions in the spleen cell cultures of leukemic animals were expressed more sharply. The relationship of LDH isozymes was not changed by treating embryonal tissue culture cells with normal or leukemic splenic extracts. (5 references)

4177 THE CAPABILITY FOR HORMONE-STIMULATED ENZYME ADAPTATION IN LIVER CELLS ISOLATED FROM AGING RATS. (Eng.) Britton, G. W. (Philadelphia Coll. Osteopathic Medicine, Philadelphia, PA); Britton, V. J.; Gold, G.; Adelman, R. C. *Exp Gerontol* 11(1/2): 1-4; 1976.

Hepatocytes from male Sprague-Dawley rats, aged 2, 12, and 24 mo, were studied in vitro for tyrosine aminotransferase adaptation to stimulation by various concentrations of hydrocortisone. The time course, magnitude of response, and sensitivity to hydrocortisone were identical in all rats. These results suggest that the age-dependent lesions in hepatic enzyme adaptation are not intrinsic to the liver. It is possible that an endocrine disorder is responsible for these lesions.

4178 HIGH MOLECULAR WEIGHT DEOXYRIBONUCLEIC ACID POLYMERASE FROM CROWN GALL TUMOR CELLS OF PERIWINKLE (*VINCA ROSEA*). (Eng.) Gardner, J. M. (Dept. Plant Pathology, Univ. California, Davis, CA 95616); Kado*, C. I. *Biochemistry* 15(3): 688-696; 1976.

A high molecular wt (6S) plant DNA polymerase from axenic *Vinca rosea* tissue culture cells was purified 2200-fold and characterized. The enzyme has a molecular wt of 105,000 (±5000). Sodium dodecyl sulfate-acrylamide gel electrophoresis of the purified enzyme yields polypeptide subunits having molecular wt of 70,000 and 34,000. The purified enzyme has a pH optimum of 7.5; a cation requirement optimum of 6 mM Mg^{2+} or 0.5 mM Mn^{2+}; an apparent requirement for Zn^{2+}; a Km of 1 μM for dTTP; and ınd a 3.5-fold stimulation by 50 mM KCl. The enzyme is sensitive to N-ethylmaleimide (1 mM), heparin (0.1 μM), ethanol (5%), pyrophosphate (0.05 μM), and o-phenanthroline (0.1 mM) but is insensitive to rifamycin. Denatured DNA is found to be the best natural template, and only negligible activity can be demonstrated with the ribopolymer templates poly(dT)n·poly(rA)n and $p(dT)_{10}$·poly(rA)n. In addition to the polymerization reaction, the enzyme catalyzes a pyrophosphate exchange reaction. Antibody to calf thymus 6-8S DNA polymerase does not inhibit DNA polymerase from *Vinca rosea*, suggesting no antigenic relationships between the mammalian and plant enzymes.

4179 COVALENTLY CLOSED CIRCULAR DNA MOLECULES OF LOW SUPERHELIX DENSITY AS INTERMEDIATE FORMS IN PLASMID REPLICATION. (Eng.) Timmis, K. (Dept. Medicine, Stanford Univ. Sch. Medicine, Stanford, CA 94305); Cabello, F.; Cohen, S. N. *Nature* 261: 512-516; 1976.

Evidence is presented that the constructed recombinant pSC134 plasmid of *Escherichia coli* replicates via a covalently closed circular DNA species with a reduced number of supercoils. This species is termed heavy replicative DNA (hrDNA). It was revealed as a minor heavy peak on cesium chloride-propidium diiodide equilibrium centrifugation gradients following pulse-labeling of replicating DNA with ^{3}H-thymidine and a 60-sec chase. With chases lasting more than 60 sec, the amount of hrDNA progressively decreased until it disappeared after 420 sec. These kinetics suggest that it represents a plasmid replicative intermediate, appearing transiently before conversion to form I DNA. The buoyant density of hrDNA was heterogeneous. During the chase period, the amount of a heavier hrDNA component decreased from 7% of the total ^{3}H label after a 60-sec chase to 2% after a 180-sec chase and 1% after a 420-sec chase, without changing position in the gradient. In contrast, a lighter component moved progressively toward form I DNA with increasing periods of chase. Treatment with RNase demonstrated that hrDNA contains an RNA component of unknown function calculated to represent about 1% of the DNA. Electron microscopy showed that most of the molecules of hrDNA are completely untwisted. The finding that hrDNA, a closed circular monomeric DNA lacking supercoils, is an intermediate in plasmid replication suggests that newly separated progeny DNA molecules may be sealed to a closed circular form before maturation involving the introduction of superhelical turns. (34 references)

4180 SEQUENCE HOMOLOGIES IN MAMMALIAN 5.8S RIBOSOMAL RNA. (Eng.) Nazar, R. N. (Tumor By-Products Lab., Baylor Coll. Medicine, Houston, TX 77025); Sitz, T. O.; Busch, H. *Biochemistry* 15(3): 505-508; 1976.

To determine the extent of homology among mammalian 5.8S rRNAs and to detect evolutionary drifts, 5.8S rRNAs from mouse, human cells and rat cells were compared. Oligonucleotides were analyzed for pseudouridylic acid by alkaline digestion followed by electrophoresis on Whatman 3 MM paper and ascending chromatography on cellulose thin layers in propan-2-ol (680 ml)-HCl (176 ml)-H_2O (to 1l). Oligonucleotide products of complete pancreatic or T_1 RNase digestion or partial T_1 RNase digestion of HeLa cell (human) and MPC-11 cell (mouse) 5.8S rRNA are identical with those obtained from Novikoff hepatoma (rat) 5.8S rRNA except for minor differences at the termini. pCp is the only major 5' terminus of both human and mouse RNAs; both pGp and pCp 5' termini were found in rat 5.8S RNA. Furthermore, HeLa cells contain C-U-U at the 3' end rather than the cytosine-uridine terminus of mouse and rat. The results indicate that the nucleotide sequence has been highly conserved during the evolution of mammals and suggest that this sequence is essentially constant throughout the Mammalia.

4181 MALIGNANCY OF HYBRIDS DERIVED FROM IN VIVO FUSION OF TUMOR CELLS WITH HOST CELLS (Meeting Abstract). (Eng.) Jami, J. (Institut de Recherches Scientifiques sur le Cancer, Villejuif, France); Aviles, D. *J Cell Biol* 70(2/Part 2): 34a; 1976.

4182 MALIGNANT CHARACTERISTICS OF A HUMAN BREAST TUMOR LINE AFTER LONG-TERM IN VITRO CULTIVATION (Abstract). (Eng.) Miller, E. E. (Univ. Pennsylvania Sch. Medicine, Philadelphia, PA 19174). *In Vitro* 12(4): 331; 1976.

4183 MORPHOLOGICAL, BIOCHEMICAL AND CHROMOSOMAL CHARACTERIZATION OF BREAST TUMOR LINES FROM PLEURAL EFFUSIONS (Abstract). (Eng.) Cailleau, R. (M. D. Anderson Hosp. and Tumor Inst., Houston, TX 77025); Cruciger, Q.; Hokanson, K. M.; Olive, M.; Blumenschein, G. *In Vitro* 12(4): 331; 1976.

4184 CHARACTERIZATION OF T98: A POLYPLOID HUMAN TUMOR CELL LINE SHOWING NORMAL GROWTH REGULATION IN VITRO (Meeting Abstract). (Eng.) Stein, G. H. (Dept. Molecular, Cellular, Developmental Biology, Univ. Colorado, Boulder, CO 80302). *J Cell Biol* 70(2/Part 2): 24a; 1976.

4185 SCANNING ELECTRON MICROSCOPIC OBSERVATIONS ON CELLS GROWN IN VITRO. III. THE UNDERSIDE OF HELA CELLS. (Eng.) Liebrich, W. (Deutches Krebsforschungszentrum, D69, Heidelberg, P.O. Box 101949, W. Germany); Paweletz, N. *Cytobiologie* 12(2): 321-331; 1976.

4186 ALTERATIONS IN MITOCHONDRIAL MEMBRANE STRUCTURE OF MORRIS HEPATOMA 7777 (Meeting Abstract). (Eng.) Waite, M. (Bowman Gray Sch. Medicine, Winston-Salem, NC); Cunningham, C. C.; Jester, R.; Miller, N. *IRCS Med Sci (Suppl)* 3(10): 3; 1976.

4187 THREE-DIMENSIONAL STRUCTURE OF EHRLICH TUMOR MITOCHONDRIA DURING RESPIRATION (Meeting Abstract). (Eng.) Martin, A. P. (Dept. Pathology, Univ. Missouri, Columbia, MO 65201); Koukl, J. F.; Vorbeck, M. L. *J Cell Biol* 70(2/Part 2): 36a; 1976.

4188 GENETIC MOSAICISM AND THE CONTROL OF CANCER. (Eng.) Moolten, F. L. (Cancer Res. Center and Dept. Microbiology, Boston Univ. Sch. Medicine, 80 E. Concord St., Boston, MA 02118). *Med Hypotheses* 2(3): 79-81; 1976.

4189 IS REPRESSION OF TUMOR FORMATION BY CELL MIXTURES A REFLECTION OF PLASMINOGEN ACTIVATOR SUPPRESSION? (Meeting Abstract). (Eng.) Newcomb, E. (Cornell Univ. Medical Coll., Mt. Sinai Sch. Medicine, Rockefeller Univ., New York, NY 110021); Silagi, S.; Christman, J.; Kyner, D.; Acs, G.; Silverstein, S. C. *J Cell Biol* 70(2/Part 2): 129a; 1976.

4190 A MODEL IMPLICATING THE LANGERHANS CELL IN KERATINOCYTE PROLIFERATION CONTROL. (Eng.) Potten, C. S. (Christie Hosp. and Holt Radium Inst., Manchester M20 9BX, England); Allen, T. D. *Differentiation* 5(1); 43-47; 1976.

4191 IMPROVED METHODS FOR REDUCING CALCIUM AND MAGNESIUM CONCENTRATIONS IN TISSUE CULTURE MEDIUM: APPLICATION TO STUDIES OF LYMPHOBLAST PROLIFERATION IN VITRO. (Eng.) Brennan, J. K. (Depts. Medicine and Radiation Biology/Biophysics, Univ. Rochester Sch. Medicine, Rochester, NY 14642); Mansky, J.; Roberts, G.; Lichtman, M. A. *In Vitro* 11(6): 354-360; 1975.

4192 UNBALANCE OF THE SERUM COPPER AND IRON LEVELS IN BRONCHOPULMONARY CANCERS AND SERIOUS LUNG TUBERCULOSES. (Fre.) Le Tinier, J.-Y. (Services de Biochimie et de Pneumophtisiologie, C.H.U. de Paris XIII, Hopital Franco-Musulman, F 93000 Bobigny, France); Burnichon, J.; Garnier, J.-M.; Pre, J. *Nouv Presse Med* 5(10): 649; 1976.

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PREFACE

Carcinogenesis Abstracts is a publication of the National Cancer Institute. The journal serves as a vehicle through which current documentation of carcinogenesis research highlights are compiled, condensed, and disseminated on a regular basis. It represents an integral part of the Institute's program of fostering and supporting coordinated research into cancer etiology. Issues of *Carcinogenesis Abstracts* normally contain three-hundred abstracts and three-hundred citations (unaccompanied by corresponding abstracts). Abstracts and citations refer to the current scientific literature that describes the most significant carcinogenesis research carried on at the National Cancer Institute, other governmental agencies, and private institutions. *Carcinogenesis Abstracts* is intended to be a highly useful current awareness tool for scientists engaged in carcinogenesis research or related areas. The great number and diversity of publications relevant to carcinogenesis make imperative the availability of this service to investigators whose work requires that they keep abreast with current developments in the field.

Carcinogenesis Abstracts is normally published monthly. Volume XIV covers the scientific literature published from Jan 1976 through Dec. 1976. To increase the usefulness of *Carcinogenesis Abstracts*, Volume XIV, a Wiswesser Line Notation index and a Chemical Abstracts Service Registry Number index have been provided. These indexes reference compounds described in abstracted articles. A cumulative subject, author, CAS Registry Number, and Wiswesser Line Notation index for Volume XIV will be published shortly after the final regular issue.

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NOTE

Journal names are abbreviated according to the list of abbreviations used by *Index Medicus*. For journals not covered by *Index Medicus*, the abbreviations found in *Chemical Abstracts Service Source Index*, 1907-1974 Cumulative, are used. New journals are verified in *New Serial Titles* and abbreviated according to *International Standard ISO 833*. An asterisk indicates the author to address (other than the primary) in requesting reprints.

LANGUAGE ABBREVIATIONS

Afr.	Afrikaans	Ind.	Indonesian
Ara.	Arabic	Ita.	Italian
Bul.	Bulgarian	Jpn.	Japanese
Chi.	Chinese	Kor.	Korean
Cro.	Croation	Lav.	Latvian
Cze.	Czech	Lit.	Lithuanian
Dan.	Danish	Nor.	Norwegian
Dut.	Dutch	Pol.	Polish
Eng.	English	Por.	Portuguese
Est.	Estonian	Rum.	Rumanian
Fin.	Finnish	Rus.	Russian
Fle.	Flemish	Ser.	Serbo-Croatian
Fre.	French	Slo.	Slovak
Geo.	Georgian	Spa.	Spanish
Ger.	German	Swe.	Swedish
Gre.	Greek	Tha.	Thai
Heb.	Hebrew	Tur.	Turkish
Hun.	Hungarian	Ukr.	Ukrainian
Ice.	Icelandic	Vie.	Vietnamese

ABBREVIATIONS USED IN ABSTRACTS

A	angstrom(s)	µM	micromolar
ACTH	adrenocorticotropic hormone	mOsm	milliosmolar
ADP	adenosine diphosphate	max	maximum
AMP	adenosine monophosphate	mEq	milliequivalent(s)
ATP	adenosine triphosphate	min	minute(s)
approx	approximately	ml	milliliter
av	average	µl	microliter(s)
BCG	bacillus Calmette-Guerin	mm	millimeter(s)
bid	twice daily	mo	month(s)
C	degree(s) centigrade	N	normal concentration
cal	calorie(s)	NAD	nicotinamide adenine dinucleotide
kcal	kilocalorie(s)	NADH	reduced nicotinamide adenine dinucleotide
cc	cubic centimeter(s)	NADP	nicotinamide adenine dinucleotidephosphate
Ci	curie(s)	NADPH	reduced nicotinamide adenine dinucleotide-phosphate
mCi	millicurie(s)		
µCi	microcurie(s)	NCI	National Cancer Institute
cm	centimeter(s)	NIH	National Institutes of Health
CNS	central nervous system	PAS	periodic acid--Schiff
cpm	counts per minute	po	orally
DNA	deoxyribonucleic acid	ppb	parts per billion
ED_{50}	median effective dose	ppm	parts per million
EDTA	ethylenediamine tetraacetic acid	R	roentgen
g	gram(s)	RBC	red blood cells (erythrocytes)
kg	kilogram(s)	RNA	ribonucleic acid
mg	milligram(s)	rpm	revolutions per minute
µg	microgram(s)	sc	subcutaneous
Hb	hemoglobin	sec	second(s)
hr	hour(s)	SGOT	serum glutamic-oxalacetic transaminase
ia	intra-arterial	SGPT	serum glutamic-pyruvic transaminase
id	intradermal	soln	solution
ILS	increased life span	TCD	tissue culture dose
im	intramuscular	TCD_{50}	median tissue culture dose
ip	intraperitoneal	tid	three times daily
IU	International Unit(s)	UV	ultraviolet
iv	intravenous	WBC	white blood cells (leukocytes)
Km	Michaelis constant	wk	week(s)
LD	lethal dose	wt	weight
LD_{50}	median lethal dose	x	times
M	molar	yr	year(s)

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4201 A STUDY OF CARCINOGENIC MECHANISMS RELEVANT TO PUBLIC HEALTH PROPHYLAXIS. (Ger.) Wettig, K. (Hygiene-Institut, 108 Berlin, Otto-Grotewohl-Strasse 1, East Germany); Obrikat, H. *Z Gesamte Hyg* 21(10): 736-738; 1975.

A review of evidence that implicates environmental factors in raising the risk of human cancer is presented. Particular attention is given to the ambiguities in current data and their interpretation. For example, is the deceleration of hepatic 3,4-benzpyrene degradation by air pollutants a carcinogenic or carcinolytic effect? Development of a prescreening test system using yeast cells (*S. cerevisiae*) is described in which both proliferation rate and anaerobic fermentation are used as measures of toxicity by carcinogenic hydrocarbons. An early result shows that hydrocarbon mixtures elicit a much higher metabolic response than hydrocarbons separately. (7 references)

4202 METABOLIC HOST FACTORS IN CARCINOGENESIS. (Eng.) Saffiotti, U. *In: Host Environment Interactions in the Etiology of Cancer in Man. Proceedings of a Meeting Held at Primosten, Yugoslavia, 27 August-2 September 1972.* Edited by R. Doll, I. Vodopisa, and W. Davis. International Agency for Research on Cancer. (Lyon, France): pp. 243-252; 1973.

The sequence of events through which an exogenous chemical carcinogen must pass before inducing neoplastic transformation is reviewed, with emphasis on metabolic host factors. For penetration into the whole organism, the route and mode of exposure are determining factors but the conditions of the host-barrier tissues can be equally important. Molecular logistics, such as the diffusion of a chemical in the body, its distribution to various organs, its retention rates, and its elimination, also influence carcinogenic expression. The metabolism of most carcinogens appears to lead to the formation of polar noncarcinogenic compounds (detoxification). Some metabolic reactions, however, result in the formation of metabolites with carcinogenic activities equal to or greater than that of the compound administered. For those carcinogens that require metabolic conversion to a highly reactive form, capable of binding to target macromolecules, the final step appears to take place directly within the target cells. Little is known about the regulation of membrane activities that control the intake of carcinogens into target cells. Expression of transformation, even after the interactions of the carcinogen inside the cell, is still subject to regulation by metabolic processes. It is postulated that regulation occurs at the level of the cell membrane, which determines the rate of intake of critical factors into the cell that determine neoplastic expression. Attempts to develop a suitable animal model for the study of metabolic host factors in respiratory carcinogenesis resulted in a system using the Syrian golden hamster and a polynuclear hydrocarbon in a fine particulate suspension mixed with particles of a carrier dust, administered by intratracheal instillation. Recent studies revealed the critical function of vitamin A in the control of epithelial differentiation, its role in the control of basic functions in epithelial cells, and its inhibition of carcinogenesis in the respiratory tract, in the gastrointestinal tract and in the cervix. Similar mechanisms may be involved in determining susceptibility to carcinogenesis in many target tissues. (39 references)

4203 AN OVERVIEW OF CARCINOGENESIS AND MUTAGENESIS PROGRAMMES OF THE NATIONAL CENTER FOR TOXICOLOGICAL RESEARCH. (Eng.) Fishbein, L. *In: Screening Tests in Chemical Carcinogenesis. Meeting Held in Brussels, 9-12 June 1975.* International Agency for Res. on Cancer. (Brussels, Belgium): no. 12, pp. 41-57, 59; 1976.

The major ongoing carcinogenesis programs at the National Center for Toxicological Research are reviewed. The primary objective of the chronic low dose-response ED_{01} study with 2-acetylaminofluorene (2-AAF, 0, 100, 250, and 500 ppm) is to establish a quantitative experimental base for evaluating excessive carcinogenic risk produced by a chemical in Balb/c mice; these data should help determine populations at risk. Age-sensitivity studies are designed to detect possible differences in susceptibility to 2-AAF induction of urinary bladder tumors at three life periods in BALB/c female mice. The data indicate that the latent period for bladder tumor development is apparently a function of age, with the highest incidence in animals treated from 6 to 12 mo and sacrificed 3 mo later. Extensive metabolic and chronic studies with 2-AAF, benzidine, and congeners are underway to determine the carcinogenic potential of various metabolites, the binding patterns of metabolites to tissues, and the application of pharmacokinetics to carcinogenesis. The carcinogenesis bioassay program for the evaluation of rodent hepatoma may help to assess chemical carcinogens, correlate similarities between hepatomas produced by chemical carcinogens and toxic agents, and determine the influence of dose level, route of administration, and age on hepatomas. Other ongoing research programs include the etiology of bladder cancer in animals and man, the carcinogenic potential of estrogenic activity, the risk of tumor production from exposure in utero to environmental chemicals, and the role of DNA repair in carcinogenesis. (22 references)

4204 OCHRATOXIN A. (Eng.) IARC Working Group. *In: IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Man: Some Naturally Occurring Substances. Meeting Held in Lyon, 7-13 October 1975.* International Agency for Res. on Cancer. (Lyon, France): vol. 10, pp. 191-197; 1976.

The chemical, physical, and carcinogenic properties of ochratoxin A were reviewed. Oral administration of 100 or 300 µg/rat (males and females) for 5 days/wk for 50 wk resulted in one hamartoma of the kidney in the animal receiving the highest dose. Rainbow trout did not develop tumors when fed ochratoxin A for 8 mo at 16, 32, or 64 µg/kg of diet, but hepatomas were produced when sterculic acid was included in the diet. No tumors were observed in 7/20 survivors

81 wk after sc injection of 10 male and 10 female mice with 10 μg ochratoxin A in 0.1 ml arachis oil twice weekly for 36 wk. Local fibrosarcomas developed in 2/10 female Wistar rats given sc injections of 2.5 mg/kg body weight ochratoxin A in sunflower seed oil weekly for 18 wk; 2/10 controls (sunflower oil alone) also developed local fibrosarcomas. Acute oral LD_{50}'s of ochratoxin A for rats were 22 mg/kg for males and 20 mg/kg for females. Nephropathy has been observed experimentally in pigs, dogs, rats, chickens, and trout. Increased prenatal mortality, decreased fetal wt, and fetal malformations were manifested by pregnant mice given one dose ip on days 7-12 of gestation. (18 references)

4205 MITOMYCIN C. (Eng.) IARC Working Group. *In: IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Man: Some Naturally Occurring Substances. Meeting Held in Lyon, 7-13 October 1975.* International Agency for Res. on Cancer. (Lyon, France): vol. 10, pp. 171-179; 1976.

The chemical, physical, and carcinogenic properties of the antibiotic mitomycin C were reviewed. Strain differences were observed in tumor production in mice after 35 twice-weekly injections of 0.2 μg mitomycin C in saline--all btK mice and 2/10 C57Bl mice developed local sarcomas, but no tumors developed in C3H, ddO, or control mice. Peritoneal sarcomas developed in 27/29 male and 30/31 female rats given ip injections of 0.038 or 0.15 mg/kg body wt three times weekly for 6 mo. A group of 96 BR46 male rats given five iv injections of 0.52 mg/kg mitomycin C within a 2-wk period and observed for their life span developed malignant tumors of various types in 27/79 animals and benign tumors in 3/79 animals. Controls exhibited 4/65 malignant tumors. The LD_{50} dose (single ip dose) for rats was 2.5 mg/kg and for mice 8.5 mg/kg. The oral LD_{50}'s were 50 mg/kg in mice and 30 mg/kg in rats. The iv LD_{50}'s were 5 mg/kg in mice and 1-2.5 mg/kg in cats, dogs, and monkeys. Mitomycin C induces genetic damage or alteration in *Salmonella typhimurium, Saccharomyces cerevisiae, Ustilago maydis, Glycine max, Drosophila melanogaster, Habrobracon*, mouse spermatogonia, and human peripheral leucocyte cultures. (33 references)

4206 NATIVE CARRAGEENANS. (Eng.) IARC Working Group. *In: IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Man: Some Naturally Occurring Substances. Meeting Held in Lyon, 7-13 October 1975.* International Agency for Res. on Cancer. (Lyon, France): vol 10, pp. 181-190; 1976.

The chemical, physical, and carcinogenic properties of the carrageenans (Burtonite V-40-E, Galozone, Pellugel, Viscarin) were reviewed. Groups of five male and five female mice fed diets containing 1%, 5%, or 25% food-grade native carrageenan exhibited no mortality effects or no apparent pathological effects in the gastrointestinal tract, liver, or kidneys. Similar results were observed in rats, but in animals fed 25% carrageenan some cases of hepatic cirrhosis were observed. Female Wistar rats developed masses of new fibrous tissue at the site of sc injections of 5 ml of 1% wt/vol carrageenan in 0.9% saline. Sarcomas developed in 11/39 animals reinjected at the same site over a period of 400 days. Rhesus monkeys given 1% native carrageenan in drinking water for 7-14 wk had no adverse effects; however, 1% degraded carrageenan produced wt loss, hemorrhages in the intestinal tract, anemia, mucosal erosion leading to ulceration, and formation of granulation tissue in the lamina propria and multiple crypt abscesses. Ulcerative lesions were observed in guinea pigs and rats receiving a 5% solution of native or a 1% solution of degraded carrageenan in drinking water. (20 references)

4207 COUMARIN. (Eng.) IARC Working Group. *In: IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Man: Some Naturally Occurring Substances. Meeting Held in Lyon, 7-13 October 1975.* International Agency for Res. on Cancer. (Lyon, France): vol. 10, pp. 113-119; 1976.

The chemical, physical, and carcinogenic properties of coumarin were reviewed. Bile duct proliferation, cholangiofibrosis, and focal necrosis were observed in Osborne-Mendel rats of both sexes fed 2,500 or 5,000 mg coumarin/kg diet (containing 3% corn oil) for 2 yr. In a separate study of 24/40 albino rats fed 5,000 mg/kg diet for 2 yr and surviving 18 mo or more, 11 males and 1 female developed bile duct carcinomas. In a subsidiary study including 82 rats, 4/25 males and 1/8 females surviving 18 mo or more and fed 3,500 mg/kg diet developed bile duct carcinomas. Rats fed 1,000 and 2,500 mg/kg of diet developed a few adenomas of the bile ducts, but none were found in controls. The LD_{50}'s in mice were 196 mg/kg body wt po and 310 mg/kg sc; in rats fed a 5% solution in corn oil, 680 mg/kg; and in guinea pigs fed a 10% solution in polypropylene glycol, 200 mg/kg. In man 68%-92% of an oral dose of coumarin is excreted in the urine as 7-hydroxycoumarin and 1%-6% as 2-hydroxyphenylacetic acid. Coumarin and some of its metabolites inhibit liver glucose-6-phosphatase, and coumarin itself interferes with excision repair processes in UV damaged DNA. (17 references)

4208 DAUNOMYCIN. (Eng.) IARC Working Group. *In: IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Man: Some Naturally Occurring Substances. Meeting Held in Lyon, 7-13 October 1975.* International Agency for Res. on Cancer. (Lyon, France): vol. 10, pp. 145-152; 1976.

The chemical, physical, and carcinogenic properties of daunomycin (Cerubidine, Daunorubicin, Rubomycin C) were reviewed. The incidence of reticulosarcoma or leukemia in mice (10 male and 20 female strain C57Bl/Rho) surviving 16-22 mo after receiving 12.5 mg/kg body wt daunomycin for 22 wk was 4/19 in treated mice and 12/35 in controls. Incidences of other tumors were similar in control and treated mice. Five male and five female mice (XVII/Rho) were found to have local sarcomas 22 mo after receiving 1.25 mg/kg daunomycin sc for 12 wk. Of 25 female

Sprague-Dawley rats given a single iv injection of 12.5 mg/kg, 14 rats died the following year (2 with malignant tumors), and all remaining rats killed at 12 mo had tumors (11 mammary adenocarcinomas and 2 fibroadenomas) with no tumors visible in 25 control rats. Groups of 20 Sprague-Dawley rats were given single iv injections of 5, 10, or 20 mg/kg daunomycin or saline (controls) and observed for 12 mo. All receiving the highest dose died or developed glomerulonephritis before 52 days. In the other groups of treated rats, 16/32 developed a total of 27 tumors, including 5 tubular adenomas and 2 clear-cell carcinomas of the kidney compared with 5/20 controls. The LD_{50}'s of daunomycin in GS mice were 5.6 mg/kg ip and 47.0 mg/kg sc, with considerable variation in toxicity. Doses of 12-50 mg/kg caused bradycardia, hypotension, ventricular arrhythmias, and respiratory depression in hamsters and rhesus monkeys; immunosuppression in mice; and nausea, vomiting, leucopenia, cardiac toxicity, and fatal cardiac function disturbances in man. Daunomycin complexes strongly with DNA, inhibits DNA and RNA polymerases in vitro, does not induce unscheduled DNA synthesis in cultured human fibroblasts, and induces high frequencies of chromosome and chromatid breaks and translocations in human peripheral leucocyte cultures. (26 references)

4209 MESOTHELIOMA AND ITS RELATIONSHIP TO ASBESTOS EXPOSURE. (Jpn.) Watanabe, S. (Natl. Cancer Center, Tokyo, Japan). *Jpn J Chest Dis* 35(1): 12-18; 1976.

This review paper deals with the definition and microscopic and histologic features, particularly the histochemistry, of mesotheliomas. The correlation to asbestos exposure, obtained chiefly from epidemiological considerations and animal experiments, is discussed. Mesotheliomas in which exposure to asbestos through the respiratory tract is thought to be the single carcinogenic factor are distinguished from most asbestos-related lung cancers, in which other carcinogenic factors are also involved. (17 references)

4210 CHENODEOXYCHOLIC ACID AND INTESTINAL CANCER (Letter to Editor). (Fre.) Martin, M.-S. (Laboratoire d'Immunologie et de Medecine experimentale, INSERM U-45, Faculte de Medecine, boulevard Jeanne-d'Arc, F 21000 Dijon, France); Martin, F. *Arch Fr Mal App Dig* 65(1): 60; 1976.

The long-term use of chenodeoxycholic acid, which is used to treat biliary lithiasis, may be carcinogenic in the intestine. Biliary acids or their breakdown products seem to contribute to the pathogeny of colon and rectal cancers. In humans, biliary acid excretion is significantly higher among ethnic groups with the highest incidence of intestinal cancer. Several studies have noted an increase in biliary acids in the stools of patients with colorectal tumors. Animal experiments also incriminate biliary acids. Lithocholic acid, singly, does not cause intestinal tumors in rats. When it is associated with a known carcinogen (methylnitronitrosoguanidine), the incidence rises by 110%. This result is obtained with a dose of 5 mg/wk over 11 mo. The acid, which is almost entirely eliminated through the stools, originates from the breakdown of chenodeoxycholic acid by intestinal bacteria. In the same manner, the addition to the normal rodent diet of 2% cholestyramine doubles the number of intestinal tumors in rats exposed to three different carcinogens (dimethylhydrazine, nitroxymethane, methylnitroxymethanol). The risk of a significant increase in an already prevalent cancer seems to warrant a thorough experimental study of the carcinogenic (and cocarcinogenic) effects of chenodeoxycholic acid. The only alternative would be the systematic registration of treated cases and paired controls to see if, within 10 to 20 yr, the incidence of colorectal cancer was or was not identical in the two groups.

4211 ENDOGENOUS HORMONES AND PROGNOSIS. (Eng.) Miller, H. *In: Risk Factors in Breast Cancer*. Stoll, B. A., ed. (Chicago: William Heinemann Medical Books): New Aspects of Breast Cancer, vol. 2, pp. 149-163; 1976.

It is established that estrogens initiate mammary carcinogenesis in the mouse, that oophorectomy influences the course of established human breast cancer, and that the result of hormone ablative surgery for advanced cases can be predicted with some degree of accuracy. As a result, the possibility of estrogens, and perhaps of other hormones, as initiating or modifying factors in the etiology of human breast cancer has received serious attention. Using urinary steroid excretion ''discriminatory functions,'' it has been shown that only 5% of patients who have a negative discriminant and a ''free period'' of less than 2 yr, or are within 6 yr of achieving the menopause, will benefit from hormone therapy. Premenopausal patients with negative discriminants are unlikely to benefit from adrenalectomy or hypophysectomy, but postmenopausal patients with positive discriminants are worthy of consideration for these operations. The steroid excretion changes found in breast cancer patients are believed to reflect the general systemic nonspecific metabolic manifestations of the disease. (54 references)

4212 MECHANISM OF ANDROGEN ACTION AT THE CELLULAR LEVEL. (Eng.) Sluyser, M. (The Netherlands Cancer Inst., Sarphatistraat 108, Amsterdam, The Netherlands); Kassenaar, A. A. H. *Pharmacol Ther Part B* 1(2): 179-188; 1975.

The major steps in the mechanism of action of androgens are reviewed. In both rats and humans, injected testosterone is rapidly taken up by the prostate and at least 90% is then converted to androstandiol, androsterone, and 5α-dihydrotestosterone (DHT). Prostatic nuclei reduce testosterone to DHT, and in the cytoplasm both DHT and androstandiol are formed. Receptors for DHT have been detected in the nuclei and cytosol of rat prostates, and androgen receptors have been identified in the accessory sexual glands and certain experimental androgen-dependent tumors in several species. No receptors have been found in most types of skeletal muscle. In one study, samples of hypertrophied human prostate gland were shown to contain a large amount of DHT, but in another DHT receptors were not detected in all samples of hypertrophied human prostate studied. The hypothalamus appears to be a target tissue for androgens. In the absence of cytosol, purified prostatic nuclei alone were unable to bind or retain DHT to any appreciable extent, suggesting that cytoplasmic DHT receptors are implicated in the nuclear binding of DHT. DHT receptors appear to bind specifically to DNA obtained from the prostate by the detergent method. DHT receptors stimulate RNA polymerase activity in the nuclei of androgen-sensitive cells; they do not enhance the cytoplasmic form of DNA polymerase. It is unclear how the binding of androgen-receptor complexes to nuclear chromatin causes the nuclei to produce increased amounts of RNA. Testosterone mainly induces the synthesis of ribosomal RNA and also increases the activity of seminal-vesicular phosphofructokinase. Androgens control the expression of liver N'-methylnicotinamide oxidase and affect mitochondrial protein synthesis in the rat prostate. (81 references)

4213 CONSIDERATION OF STEROID METABOLISM IN BREAST CANCER. (Eng.) Lipsett, M. B. *In: Host Environment Interactions in the Etiology of Cancer in Man. Proceedings of a Meeting Held at Primosten, Yugoslavia, 27 August-2 September 1972.* International Agency for Res. on Cancer (Lyon, France): pp. 105-113; 1973.

Steroid metabolism is discussed in relation to breast cancer. A general review of some features of steroid metabolism is followed by a discussion of the origin of ketosteroid metabolites in urine, the origin and rate of estrogen production in women, and problems of estrogen activity in pregnancy. The use of urinary metabolite measurements to obtain information about the hormonal milieu is discouraged. The hypothesis that the protection against breast cancer afforded by early pregnancy is due to the characteristically high estriol production in pregnancy is an example of confusion between estriol conjugates in urine and free estriol in blood. During pregnancy, plasma estradiol concentrations are eight times those of estriol, and estradiol is the most abundant free estrogen in blood. Estriol conjugate secretion in the urine during pregnancy is high compared to that of estradiol and estrone. Urinary metabolite measurements may have empirical value, however, as alterations of steroid metabolism may be associated with cancer. (29 references)

4214 PROTEIN AND POLYPEPTIDE HORMONES IN HUMAN NEOPLASIA. (Eng.) Ross, G. T. *In: Host Environment Interactions in the Etiology of Cancer in Man. Proceedings of a Meeting Held at Primosten, Yugoslavia, 27 August-2 September 1972.* International Agency for Res. on Cancer (Lyon, France): pp. 115-129; 1973.

Problems in measuring protein and polypeptide hormone concentrations in small volumes of human blood are discussed, the usefulness of these methods is illustrated, and suggestions are made to facilitate the examination of large numbers of samples collected from patients either at risk for or having tumors. Radioimmunoassays for protein and polypeptide hormones may be applied profitably to epidemiological studies of the biological fluids of patients with neoplasms. A suitable assay for large-scale studies should be specific, sensitive, precise, reproducible, and easily applicable. When possible, participating laboratories should agree on a single assay procedure. In addition, appropriate sampling procedures should be selected: a random sample is useful for tumor-specific hormone studies (such as human chorionic gonadotropin), but for fluctuating hormones such as follicle stimulating hormone, specimens probably should be collected during a standardized stimulation or inhibition test to eliminate the influence of cyclical variation. The results of protein and polypeptide hormone assays suggest two avenues for exploration: the development of more sensitive methods to measure hormones such as human chorionic somatomammotropin (which, under suitable conditions, may be regarded as tumor markers) to aid in early neoplasm recognition among high risk groups; and (b) the production of specific reagents to identify immunologically active (but biologically inactive) hormone varieties, which might lead to the discovery of tumor markers not yet recognized. (33 references)

4215 ANDROGEN ANTAGONISTS. (Eng.) Lerner, L. J. (Dept. Endocrinology, Lepetit Res. Labs., Gruppo Lepetit S.p.A., Milan, Italy). *Pharmacol Ther Part B* 1(2): 217-231; 1975.

Androgen antagonists--those agents that interfere directly with the effect of endogenous or exogenous androgen upon androgen-responsive tissues--are discussed. Most androgen assays are based on its effect on chick or capon comb growth or on its effect on accessory sex organ growth in the castrate rat or mouse. Among the compounds that show antiandrogenic activity are corticoids, estrogens, progestogens, compounds with weak androgen activity, and nonhormonally active compounds. The antiandrogenic activity of several corticoids in comb growth assays did not parallel their glucocorticoid or anti-inflammatory activity. A number of steroidal and nonsteroidal estrogens show antiandrogenic activity by the chick comb method but not by the castrate rat method, possibly because the activity in the second case is masked by the estrogen's trophic effect on the accessory sex organs. Progestational compounds are not universally antiandrogenic in the castrate rat, and many can interfere with secretory as well as growth responses to androgen administration. Compounds having weak androgenic activity can inhibit the effects of stronger androgens on selected target tissues. Contraction of the six-membered A, B, or C rings of androgenic steroids to five-membered rings has led to a variety of such compounds. Among the nonhormonally active antagonists are 11α-hydroxyprogesterone, 6α-bromo-17β-hydroxy-17α-methyl-4-oxa-5α-androstan-3-one, N-(3,5-dimethyl-4-isoxazolymethyl)-phthalimide, 4'-nitro-3'-trifluoromethylisobutyranilide, Δ-testololactone, and A-norprogesterone, most of which are devoid of androgenic, estrogenic, or progestational activities and have little or no corticoid activity. A-Norprogesterone has been shown to inhibit the effect of androgen at several androgen target tissues that are also responsive to estrogen while having little or no effect upon the activity of estrogen. (56 references)

4216 BIOLOGY OF PROLACTIN. (Eng.) Cowie, A. T. (Natl. Inst. Res. Dairying, Shinfield, Reading, RG29AT, England); Forsyth, I. A. *Pharmacol Ther Part B* 1(3): 437-457; 1975.

The functions of prolactin in mammals and some other organisms are reviewed. The numbers of prolactin-secreting cells vary within and among species. Bioassay methods for prolactin include the pigeon crop sac assay, lactogenic responses to prolactin in the rabbit, luteotropic responses in the rat or mouse, and miscellaneous responses such as its ability to release β-glucuronidase activity in rat testis homogenates or to alter behavior in lower vertebrates. Radioimmunoassay methods, including radioreceptor assays and densitometric measurements, are also available. The major biologic responses to prolactin fall into five main categories: effects related to reproduction; effects on osmoregulation and the cardiovascular system; effects related to growth and metabolism; effects on integumentary or ectodermal structures; and effects related to synergism with steroids. It also appears to affect the immune system. Current concepts of the action of protein hormones on target cells envisage that the hormone first binds to specific high-affinity receptor sites on the target cell membrane, after which the hormone-receptor complex activates hormone-dependent processes in the cell. Prolactin is often oversecreted by pituitary adenomas, in patients with galactorrhea, and in response to a variety of stresses. It is generally tumorigenic, but may inhibit or retard carcinogenesis. Prolactin secretion is controlled by a prolactin-inhibiting factor, a prolactin-releasing factor, thyrotropin, hypothalamic biogenic amines, estrogens, a number of drugs, milking stimulus, its own negative feedback, nonspecific stresses, and circadian rhythms. The levels of prolactin in the plasma of man, cattle, goats, sheep, rats, and mice under varying conditions are reported. (198 references)

4217 RADIATION. (Eng.) Miller, R. W. *In: Cancer Epidemiology and Prevention. Current Concepts*. Edited by D. Schottenfeld. (Springfield: Charles C. Thomas): pp. 93-101; 1975.

Radiation induces several forms of leukemia in humans; survivors of total-body exposure from the atomic bombs in Hiroshima and Nagasaki exhibited acute leukemias and chronic myelogenous leukemia but not chronic lymphocytic leukemia. Host factors related to age determined the cell type of leukemia induced. Partial-body radiation is also leukemogenic; x-ray treatments for ankylosing spondylitis, enlarged thymus in infancy, and childhood tinea capitis induced cases of leukemia. Data are cited that attempt to demonstrate a causal relationship between in utero diagnostic radiation and an increased risk of childhood leukemia; other studies also described a linear relationship between radiation dose and cancer risk. These studies indicate that diagnostic radiation during fetal life is associated with an increase in cancer deaths under 10 yr of age, but a definite causal relationship has not yet been proved. There also seems to be greater susceptibility to cancer following childhood irradiation. Thyroid cancer may be induced by either partial-body (x-ray) or whole-body (fallout) irradiation. Inhalation of radioactive dust can induce lung cancer, but a relation between lung cancer and external radiation is less clear. The etiological relationship between irradiation and breast cancer is also not yet demonstrated. Radioisotopes can cause bone cancer, and high therapeutic or diagnostic doses of radiation (thousands of rads) have induced fibrosarcoma and cancer of the skin; cholangiomas and hemangioendotheliomas of the liver have been observed repeatedly following the use of Thorotrast in diagnostic radiotherapy. (51 references)

4218 RADIATION EFFECTS AND AGING. (Ger.) Cottier, H. (Pathologisches Institut der Universitaet Bern, Bern, Switzerland); Buerki, H.; Gautschi, J. R.; Pedrinis, E.; Luscieti, P.; Ruchti, G.; Zimmermann, A. *Verh Dtsch Ges Pathol* (59): 139-149; 1975.

A critical review of radiation trauma and its effect on aging is presented. The origin and type of ionizing radiation to which humans are exposed, late somatic effects of high-dose, short-term ionizing whole-body radiation, somatic effects of progressive radiation at low dose rates, biological effects of the transuranium elements, and a comparison between the life-threatening late effects of radiation and aging are analyzed. The incidence of malignant neoplasms in irradiated animals exceeds the level expected, assuming accelerated or premature aging in these animals. Further, rare tumors occur early in animals after whole-body radiation exposure, an outcome unknown in large control samples of the same strain. (52 references)

4219 IONIZING RADIATION-INDUCED DNA-PROTEIN CROSS-LINKING. (Eng.) Yamamoto, O. *In: Aging, Carcinogenesis, and Radiation Biology. The Role of Nucleic Acid Addition Reactions.* Smith, K. C. Smith. (New York: Plenum Press): pp. 165-192; 1976.

Evidence for the ionizing radiation-induced cross-linking of DNA with protein derives from many observations. Unextractability of DNA following irradiation was the initial observation, and the conclusion was fortified by the finding of residual protein in isolated DNA. The interaction between DNA and protein is DNA-irradiation and protein-irradiation dependent. Sedimentation distribution analysis indicates that the number of covalent DNA-protein cross-links is less than the number of DNA double strand breaks per nucleotide. Clear evidence of the covalent binding of the constituents of protein and nucleic acid, mutually and with each other, has been reported in aqueous biochemical systems. Aromatic and sulfur-containing amino acids are particularly reactive, while aliphatic amino acids show little activity. The base portion of the nucleic acid appears to be the site of binding. Oxygen inhibits these radiation-induced covalent linkages. It is concluded that the thiyl radicals from sulfur-containing amino acids and the phenyl- and phenoxyl-type radicals from aromatic amino acid residues in protein may mainly attack the 6-position in pyrimidine base rings and the 8-position in purine base rings in the nucleic acid. (76 references)

4220 TUMOR VIRUSES: 1974. (Eng.) Baltimore, D. *In: Tumor Viruses. Cold Spring Harbor Symposia on Quantitative Biology.* Cold Spring Harbor Lab. (Cold Spring Harbor, NY): vol. 39, pp. 1187-1200; 1975.

Most of what is known about the RNA tumor virus can be codified by consideration of four genetic elements. Singly, these four genes (*env, pol, gag,* and *onc*) are responsible for the production of a protein that the virus uses to absorb cells, the encoding of the precursor to all major internal antigens of the virus, or the production of the protein of a sarcoma virus that is able to transform the growth property of cells. Most evidence strongly suggests that the genome of the RNA tumor viruses is a polyploid genome consisting of at least two identical subunits. Reverse transcriptase is the enzyme thought to be responsible for synthesizing the DNA copy of viral RNA. Once the viral DNA is integrated into the cellular DNA, it becomes a template for the synthesis of viral RNA. The synthesis of all viral proteins appears to be achieved by the synthesis of a polyprotein, a single chain of amino acids that must be cleaved to yield the functional proteins. The regulation of expression of the RNA tumor virus genome is not clear. The ability of RNA tumor viruses to become established in cells as integrated proviral DNA means that a cell can acquire a viral genome either by infection or by inheritance. Evidence for each pathway is reviewed. The current information on polyoma virus, adenovirus, herpesvirus, and Epstein-Barr virus was discussed in detail, including the concept of transformation. (27 references)

4221 RNA TUMOR VIRUS GENETICS 1975. (Eng.) Vogt, P. K. (Univ. Southern California, Sch. Medicine, Los Angeles, CA). *Adv Pathobiol* 4: 29-37; 1976.

The genetics of RNA tumor viruses is reviewed. The genome of the RNA tumor virus is described in terms of its physicochemical properties. Conditional (temperature sensitive, ts) as well as nonconditional mutants of type C viruses have been isolated. Many of the latter represent deletions. Three classes of mutants are identified according to their functional defects: class T mutants lack transforming ability; class R mutants cannot synthesize infectious progeny; and class C mutants have a defect in a viral gene required for transformation and replication alike. Examples of each are cited. Recombination is common among RNA tumor viruses and was first observed in double infections with nondefective avian sarcoma and avian leukosis virus having a different envelope specificity. All markers of RNA tumor viruses appear to be able to participate in genetic recombination. Recombinants must arise by molecular crossing-over between homologous nucleic acid molecules. (8 references)

4222 TYPE C VIROGENES: GENETIC TRANSFER AND INTERSPECIES TRANSFER. (Eng.) Todaro, G. J. (Natl. Cancer Inst., Bethesda, MD 20014). *Adv Pathobiol* 4: 38-46; 1976.

Studies of the genetic and interspecies transfer of type C virogenes are reviewed. Activation of normally repressed genetically transmitted type C virogene information, rather than infection from outside the animals, was proposed as the most common mechanism by which type C RNA tumor viruses are maintained in animal populations and produce naturally occurring cancers. Implications of the virogene-oncogene hypothesis are that all somatic cells of a species have DNA homologous to type C virus RNA of that species and that type C viruses derived from closely related species should have closely related gs antigens. Normal cellular DNA should contain transformation specific sequences of RNA tumor viruses, and spontaneous, chemically induced, and viral induced transformed cells and tumor cells should have RNA as well as DNA sequences homologous to the transforming specific sequences found in tumor viruses. The major differences between the virogene and protovirus models are discussed. Hybridization studies using endogenous baboon type C viral DNA indicated that sequences related to those of the baboon type C virus are present in all other Old World monkey species, higher apes, and in man. This supports the conclusion that in the primates, type C viral genes have evolved as the species have evolved, with virogenes from more closely related genera and families showing more sequence homology than those from distantly related taxons. Viral genes from one group of animals can give rise to infectious particles that can not only integrate into the DNA of animals of other species, but can also be incorporated into the germ line. Humans, like other primates, have type C sequences in their genome, and can, in some circumstances, express at least one type C viral protein. The possible role of viruses as natural transmitters of genes between species is discussed. (16 references)

4223 GENETICS OF RESISTANCE OF FOWL TO INFECTION BY RNA TUMOUR VIRUSES. (Eng.) Pani, P. K. (Houghton Poultry Res. Station, Houghton, Huntington, PE17 E DA, England). *Proc R Soc Med* 69(1): 43-48; 1976.

The viral information of the subgroup E virus is carried endogenously by all chick cells, but only under certain conditions is this genetic information translated into a complete virus. The factors that control the complete expression of the viral genome are not yet known. One possibility could be the inhibitor Ic gene, which suppresses expression of the susceptibility es gene. Host cells have genes that control infection by the exogenous viruses of subgroups A, B, C, and D. The segregation of resistant and susceptible phenotypes in the chicken population has supported the concept that chickens carry these genes, but the mechanism of genetic control is explained by two critical assumptions. The first is that cells carry receptors on the cell surface for attachment of the tumor viruses, and the second is that these receptors are coded for by the cell genes. Nothing is known of the molecular structure of the receptors, or of why chick cells should have susceptibility genes for harmful exogenous viruses. A possible pleiotropic action of these genes, beneficial to the host, could be responsible for their maintenance in chickens. (46 references)

4224 REGULATORY GENES INFLUENCING THE RESPONSE TO ENDOGENOUS LEUKEMIA VIRUSES. (Eng.) Lilly, F. *In: Biology of Radiation Carcinogenesis*. Edited by J. J. Yuhas; R. W. Tennant; J. D. Regan. (New York: Raven Press): pp. 189-193; 1976.

The genetic features governing the AKR mouse leukemic phenotype were analyzed by a review of relevant studies; an analogy based on the classical concept of structural versus regulatory genes was presented. A cross of AKR x RF/J mice was examined for a new repressor gene. RF/J mice are very sensitive to the induction of leukemia by the percutaneous application of 3-methylcholanthrene (MCA). The low leukemia F_1 hybrids show this sensitivity as a dominant trait. It is hypothesized that the RF/J genome includes a leukemia-inducing gene but that this gene is kept in check by a repressor gene. The repressor gene is dominant in crosses and can also repress the leukemia-inducing genes of the AKR genome. The repressor gene itself is suppressed by MCA. These properties distinguish this repressor gene from both *Fv-1* and *H-2*. The hypothesis might be extended to encompass radiation leukemogenesis. (16 references)

4225 CHARACTERIZATION OF MURINE LEUKEMIA VIRUS-SPECIFIC PROTEINS. (Eng.) Fleissner, E.; Ikeda, H.; Tung, J.-S.; Vitetta, E. S.; Tress, E.; Hardy, W., Jr.; Stockert, E.; Boyse, E. A.; Pincus, T.; O'Donnell, P. *In: Tumor Viruses. Cold Spring Harbor Symposia on Quantitative Biology*. Cold Spring Harbor Lab. (Cold Spring Harbor, NY): vol. 39, pp. 1057-1066; 1975.

Mouse leukemia virus (MuLV) proteins and antigens were characterized with respect to the virion polypeptides bearing particular antigenic determinations, the biosynthesis kinetics of viral proteins during single-cycle viral growth in tissue culture, and the molecular nature of the virus-related antigens that can be expressed in the normal tissues of virus-negative mice. Gel filtration in the presence of 6 M guanidine hydrochloride revealed seven major protein species for an avian leukosis virus (MC29) and six major species for a feline, reptilian, and murine (MuLV) virus. The avian, feline, and murine viruses contained two glycoproteins that eluted at the void volume and shortly thereafter. The individual, rechromatographed MuLV protein species were used to hyperimmunize rabbits. Strong type-specific activity was found for the resulting anti-gp70 and anti-p15 sera, while p30 antiserum and antiserum against the void volume fraction displayed group-specific reactivity. The new viral antigen recognized by the latter serum may represent a distinct MuLV protein of approximately 15,000 molecular wt; the antigen has been designated p15(E). Studies of the biosynthesis kinetics of viral proteins showed that the synthesis of p30 and p15 preceded their incorporation into virus by a relatively short time; in contrast, the synthesis of the ''x'' protein in virus preparations was linear from the time of infection. The data suggest that the assembly of virions is a relatively rapid event proceeding from relatively small pools of precursors. There was a substantial rise in the G_{IX} and Gross cell-surface antigens of NIH Swiss mouse embryo fibroblasts infected with MuLV at the time of virus production. This suggests that these antigens are encoded by the MuLV genome. Study of the immunogenetics and biochemistry of the G_{IX} antigen suggests that a protein coded for by an endogenous MuLV genome may play the role of a differentiation alloantigen of thymocytes. (42 references)

4226 STUDIES ON THE EPSTEIN-BARR VIRUS GENOME AND THE EBV-DETERMINED NUCLEAR ANTIGEN IN HUMAN MALIGNANT DISEASE. (Eng.) Klein, G. *In: Tumor Viruses. Cold Spring Harbor Symposia on Quantitative Biology*. Cold Spring Harbor Lab. (Cold Spring Harbor, NY): vol. 39, pp. 783-790; 1975.

Studies of the occurrence of Epstein-Barr virus (EBV) DNA and the EBV-determined nuclear antigen (EBNA) indicate that the presence of the nuclear antigen is strictly correlated with the presence of the viral genome; that when present, the genome occurs in multiple copies per cell; and that among the various human tumors tested, only African Burkitt's lymphoma and nasopharyngeal carcinoma are associated with detectable viral genomes. Superinfection of EBV genome-carrying nonproducer cell lines revealed a correlation between sensitivity to superinfection and the inducibility of EBV-determined early antigen (EA), the appearance of which signals irreversible damage to the cell. Not all EBV isolates can induce EA in superinfection-sensitive cells, however. In contrast to virus obtained from the ''high producer'' P3HR-1 cell line, virus derived from the B95-8 marmoset line induced only EBNA but no EA in a portion of BJA-B cells (an African Burkitt's lymphoma line that lacks the EBV genome); this allows the establishment of a stable, proliferative, virus-cell relationship. The distribution of EBV genome and EBNA-positive lymphoproliferative malignancies indicates that high antibody titers against EBV do not necessarily mean that the viral genome is associated with the tumor cells. EBV infection may lead to infectious mononucleosis, in which, in all likelihood, a small fraction of the lymphocyte population is permanently converted into virus-carrying cells. However, in African Burkitt's lymphoma (but not in those outside Africa) and nasopharyngeal carcinoma, the viral genome is in the tumor itself. In the case of African Burkitt's lymphoma, transformation of normal lymphocytes into EBV-carrying cells may be essentially similar or different in normal and tumor patients; in the former case, tumor would result from a breakdown in immune surveillance. (67 references)

4227 INTEGRATION OF DNA TUMOR VIRUS GENOMES. (Eng.) Martin, M. A. (Natl. Inst. Allergy and Infectious Diseases, Natl. Insts. Health, Bethesda, MD 20014); Khoury, G. *Curr Top Microbiol Immunol* 73: 35-65; 1976.

Controlled hybridization analyses are the most direct approach for demonstrating the integration of viral DNA sequences within the host genome. Evidence for the continued presence of viral DNA in simian virus 40 (SV40) transformed cells was first obtained in virus-rescue experiments; data indicated that the induction mechanism requires fusion of the virus-free transformed cell with the permissive cell. Integrated viral genetic information can be detected by procedures utilizing separated strands of viral DNA, an analysis of DNA reassociation kinetics, or reconstruction and calibration experiments. Since infectious SV40 can be recovered from virus-free transformed cells by cocultivation or fusion with permissive African green monkey cells, the viral genome is probably present in a latent form. Findings from experiments on SV40 genome equivalents in transformed cells suggest that a single copy of SV40 DNA is sufficient to transform a cell. The presence of multiple copies of viral DNA in transformed cell lines cannot be correlated with any phenotypic difference or the amount of virus-specific RNA present. Although viral DNA can be detected in several virus-free transformed cells, the physical relationship of the viral genome to host cell DNA has not been fully resolved. Most reports concerning the relationship of papovavirus and adenovirus DNAs to cellular DNA suggest that viral DNA is integrated into the cell genome. Recent studies with temperature-sensitive mutants of SV40 suggest that a viral gene product is necessary for continued maintenance of the transformed state. Of 11 sites in SV40 DNA involved in generation of a genome-sized DNA segment, four are involved in more than a single recombinational event; molecular rearrangements accompanying the tandem triplication of SV40 DNA sequences may involve some of the same viral DNA sites that play a role in integration (103 references)

4228 PROSPECTIVE EPIDEMIOLOGICAL STUDY AND THE ASSOCIATION OF EPSTEIN-BARR VIRUS AND BURKITT'S LYMPHOMA. (Fre.) de-The, G. (Centre international de Recherche sur le Cancer, O.M.S., 150, cours Albert-Thomas, 69008 Lyon, France); Geser, A. *C R Acad Sci* [*D*] *(Paris)* 282(14): 1387-1390; 1976.

In a context of endemic malaria, there may be a cause-effect relation between Epstein-Barr virus (EBV) and Burkitt's lymphoma (BL). This tumor's evolution in children is so rapid that it implies a triggering factor, just prior to the appearance of symptoms. EBV has been found in the tumor cells; it may act as a potential cancer agent in human, in special conditions achieved through the interference of cofactors. Endemic malaria could be a cofactor that, upon breaking down the child's immunity, liberates the oncogenic power of the virus. A seroepidemiological investigation was made of the prevalence of EBV infection across several ethnic groups with varying risks for EBV-connected diseases. It revealed that primary infection by EBV happens during the second year of life in Uganda, around the fifth year in the Far East, and not until adolescence in highly developed countries. The age of primary infection may be critical in determining the risk of BL. Next, a prospective study was conducted that consisted of registering and interviewing 40,000 children aged 2 to 5 in the Western Nile region. Blood samples were taken, and there was a followup examination to detect all tumors. Twelve BLs and one Wilms' tumor have appeared so far, 7 to 20 mo after the samples were taken. Studies on pre- and post-Burkitt sera from 8/12 cases led to 3 conclusions. All children who were going to develop BL already had antibodies against EBV 7 to 20 mo before the illness. Among EBV antibodies, those directed against structure antigens were higher in candidates to BL than in controls. After appearance of the tumor, the young patients developed antibodies against the early antigens, which meant an active infection by EBV. These results, if maintained, would indicate a causal relation between EBV and BL. An improvement in hygiene, delaying the age of primoinfection, would verify the hypothesis of the link between postnatal infection and tumoral risk. A vaccine has been considered, but it presents too many problems. Another measure would be to offer the local health authorities an antimalaria chemoprophylaxis and then watch the change in incidence of BL. The Northern Mara district in Tanzania would be an excellent site, meeting all the required conditions.

4229 INTEGRATION OF VIRAL DNA INTO THE HOST GENOME. (Eng.) Doerfler, W. (Inst. Genetics, Univ. Cologne, D-5000 Koln 41, Weyertal 121, West Germany). *Curr Top Microbiol Immunol* 71: 1-78; 1975.

The integration of DNA of animal viruses into the host genome is reviewed, as are selected topics dealing with lysogeny and the temperate bacteriophages λ and Mu. In the case of phage λ, there is a phage-encoded, highly efficient recombination system that leads to the insertion of the phage DNA into the bacterial chromosome at one specific site. Bacteriophage Mu integrates its DNA into the host genome randomly and causes polar mutations at the site of insertion. Integration has been well-documented for the adenovirus and simian virus 40 (SV40) DNAs and to a lesser extent for polyoma virus DNA. Evidence has also been presented for the covalent insertion of the reverse transcripts of tumor virus RNA, particularly in Rous sarcoma, avian myeloblastosis, and murine leukemia virus systems. Complete copies of type C virus DNA have been found in the somatic cell DNA of the chicken, Chinese hamster, Syrian hamster, mouse, rat, cat, pig, and baboon. The integration site of viral genes has been localized on a specific chromosome of eukaryotic cells in only one instance: SV40 DNA can become associated with human chromosome C-7. In human cells infected with adenovirus type 12, a specific break on chromosome 17 close to the thymidine kinase locus has been reported, but there is no evidence that the site is identical with the site of integration. Integration of viral genetic material in transformed cells has been shown in SV40-transformed cells and in cells transformed by RNA tumor viruses. In most cases of virus-transformed cells, the persisting viral genes continue to be transcribed and virus-specific messenger-RNA, frequently of high molecular wt, is found associated with the polysomes, indicating that viral genes are translated in transformed cells. (482 references)

4230 SYNOPTIC REPORT: LEUKEMIA VIRUSES. (Ger.) Hehlmann, R. (Medizinische Polikinik der Universitaet, Pettenkoferstrasse 8a, D-6000 Munich 2, West Germany). *Blut* 32(5): 375-388; 1976.

Leukemia viruses, spherically particulate and with a central core (C type), consist of glycoproteins and lipids with single-stranded RNA, 1.16 g/cm^3 in density. In mice, the autosomal genes Fv-1, Fv-2, and Rgv control leukemia virus infections. Electron microscopy indicates that there are fewer viruslike particles in human leukemias than in those of animals, whereas clinical serology has shown leukemia virus antigens in human leukocytes and plasma with human and animal leukemia virus antibody tests. Reverse transcriptase, present in human leukemia virus infections, is a sure indicator, as confirmed in previous studies using synthetic templates. Molecular hybrid structures examined by centrifugation, chromatography, and specific nuclease analysis show complementary RNA or DNA sequences in tumor cell plasma and nuclei. DNA particle sedimentation analysis immediately after viral RNA detection has proved effective for determining tumor viruses, although culturing human particles still poses problems, possibly because of endogenous viruses. Viral oncogenesis studies have attempted to explain vertical transmission of virus information by RNA transcription and incorporation as DNA provirus. Trans species infections have been noted in primates and cats, and the integrated genome has protected against secondary infection by the same virus, presenting a selective advantage.

4231 SOME ASPECTS OF THE BIOLOGIC BEHAVIOR OF EPSTEIN-BARR VIRUS. (Eng.) Miller, G. (Howard Hughes Medical Inst.) *Adv Pathobiol* 5: 25-41; 1976.

In vitro and in vivo studies in man and experimental animals relating to Epstein-Barr virus (EBV)-induced lymphoreticular cell transformation are reviewed, and unresolved questions or problems are discussed. EBV causes cell transformation or immortalization, as demonstrated by the direct action of EBV in vitro to lymphocytic cells or by culture of leukocytes from the blood of normal EBV antibody-positive individuals. Three models may account for the latter phenomenon: (a) EBV may be adsorbed to some cellular element, then transferred in vitro to uninfected lymphocytes, causing their immortalization; (b) occasional resting lymphoid cells may harbor the EB genome in a nonproductive state; it is activated on transfer to cell culture to produce mature virus that transforms uninfected cells; or (c) transformed cells containing the EBV genome may be present in the peripheral blood but are only activated when placed in vitro. Morphologic transformation and increase in cell number are preceded by viral stimulation of cellular DNA synthesis. This stimulation is inhibited by specific EBV antiserum and does not occur with nontransforming EBV. Not all EBV strains transform primary lymphocytes into cell lines. With the exception of the P^{3}J-HR-1 strain from Burkitt's lymphoma (BL), the properties of cell transformation and abortive replication may be mutually exclusive. In addition, there is an indication of heterogeneity between EBV strains associated with infectious mononucleosis (IM) and BL. EBV causes lymphoproliferative disease following experimental inoculation of nonhuman primates. However, the atypical peripheral blood lymphocytes and heterophile antibody associated with IM have not been found in experimentally infected marmosets. Addition of throat washings from IM patients to primary cultures of human umbilical cord leukocytes results in the latter forming continuous lymphoblastoid cell lines. EBV, which has been demonstrated in the throat washings, appears to be responsible for this phenomenon; this further implicates EBV as the cause of IM. (28 references)

4232 ONCOGENIC VIRUSES: THE LAST TWELVE YEARS. (Eng.) Dulbecco, R. *In: Tumor Viruses. Cold Spring Harbor Symposia on Quantitative Biology.* Cold Spring Harbor Lab. (Cold Spring Harbor, NY): vol. 39, pp. 1-6; 1975.

Progress in the study of oncogenic viruses over the last 12 yr is reviewed. The subjects discussed are: (a) the lytic cycle, transformation, integration, and DNA exchange mechanisms of DNA tumor viruses; (b) the genetic aspects, defectiveness characteristics, and the natural history of RNA tumor viruses; (c) speculation concerning a transforming gene, or ''oncogene''; and (d) the transforming function of different viruses. (50 references)

4233 HERPES SIMPLEX: FROM VESICLES TO CANCER. (Eng.) Blank, H. (Univ. Miami Sch. Medicine, P.O. Box 520875, Biscayne Annex, Miami, Fl 33152). *J Dermatol (Tokyo)* 2(2): 53-67; 1975.

Current knowledge of the possible relationship between herpesviruses and cancer is reviewed. The herpesvirus has a core of DNA-protein around which is a highly structured protein capsid; surrounding the capsid are coats made of proteins and glycoproteins. The most common form of primary herpes simplex infection is a gingivostomatitis in the mouth of children, and multinucleate giant cells in the epithelium lining the vesicle of any herpes infection are typical. Antibodies to herpesvirus type 1, varicellazoster, cytomegalovirus, and Epstein-Barr virus are present in three-fourths of the population by age 15 yr, whereas antibodies to herpes virus type 2 increase with increasing age after 15 yr. Most subjects do not have any detectable primary infection. After primary infection a latent stage occurs from which the virus can be activated by various factors, including fever, sunburn, trauma, and emotional upsets. It is possible to get recurrent lesions from an exogenous source even when antibody is present; the epithelium does not appear to be protected by serum antibody. Herpesviruses 1 and 2 can be distinguished antigenically and on the basis of different DNAs. The evidence for considering type 2 as a cause of cervical carcinoma derives from histological information, epidemiological and experimental studies, and immunologic studies in women with various stages of the disease. There is a need for active antiherpes therapy, but at present there is no effective drug for parenteral or systemic therapy. Agents such as 9-β-D-arabinofuranosyl adenine, phosphonoacetic acid, 2-deoxy-D-glucose, and the interferon inducer Poly I:C have potential for topical use; however, the only measures that have been proved useful are symptomatic forms of treatment. (13 references)

4234 BURKITT'S LYMPHOMA--A HUMAN TUMOR MODEL SYSTEM FOR IMMUNOLOGICAL STUDIES. (Eng.) Gunven, P. (Dept. Tumor Biology, Royal Karolinska Inst., S-104 01 Stockholm 60, Sweden). *Biochim Biophys Acta* 417(3/4): 187-210; 1975.

The immunological aspects of Burkitt's lymphoma are reviewed and related to hypotheses concerning the etiology, diagnosis, and clinical course of the disease. Burkitt's lymphoma occurs mainly in children in parts of tropical Africa and has epidemiological and clinical features that indicate a viral etiology and a host immune response to the tumor. Epstein-Barr virus (EBV) has been identified in Burkitt's lymphoma-derived cell lines. Immunological studies have revealed the presence of antibodies to different EBV-associated antigens in all African patients with Burkitt's lymphoma. The antibodies, however, are not specific for Burkitt's lymphoma but are found in most adults all over the world, although at lower levels. Their presence cannot serve diagnostic purposes but can give prognostic information and clues to the mechanisms behind later tumor recurrences. EBV-associated antibody titers are also high in patients with nasopharyngeal carcinoma, Hodgkin's disease, and infectious mononucleosis. Burkitt's lymphoma patients differ from appropriate control groups in which some of the persons are anti-EBV seronegative, and this, together with the presence of EBV in Burkitt's lymphoma biopsies and the absence of EBV in other lymphomas, favors an etiological role of EBV in Burkitt's lymphoma. (209 references)

4235 IMMUNOLOGIC STUDIES IN WILMS' TUMORS. (Eng.) Burtin, P. *In: Wilms' Tumor.* Pochedly, C.; Miller, D., eds. (New York: John Wiley and Sons): pp. 57-62; 1976.

Immunochemical experiments on whether nephroblastomas bear tumor-specific or tumor-associated antigens were first based on the production of specific antisera in animals with nephroblastoma extracts. Several antigens present in the normal kidney were absent in nephroblastomas. Another finding was the regular absence from the nephroblastoma of feto-specific antigens, such as α-fetoprotein. However, a fetal kidney antigen was found in 2/13 nephroblastoma extracts. Because the antitumor antisera were completely absorbed by normal kidney extracts, a tumor-specific antigen could not be characterized. More recent investigations of an abnormal mucopolysaccharide in the blood and urine of three children with Wilms' tumor proved to be of little value in the diagnosis of Wilms' tumor. Another tumor-specific antigen, W (Wilms), was not found in the normal kidney, but was seen in some carcinomas of other organs. Another antigen was identified in some nephroblastoma extracts; it does not react with anti-W antiserum, nor does its specific antiserum precipitate with W antigen. The significance of these two antigens is unknown. In two antitumor immunity studies, lymphocytes were isolated from the peripheral blood of nephroblastoma-bearing patients and from controls, and the action of these cells on cultivated nephroblastoma cells was determined. Comparable results demonstrated a common, though not always present, reactivity between different nephroblastomas. It is not known whether the antigen(s) common to many, if not all, nephroblastomas and able to sensitize patients' lymphocytes is also present in the fetal kidney and is thus carcinoembryonic. In addition, it is not clear whether immunochemistry and cytotoxicity characterize the same antigens. (11 references)

4236 THE PATHOGENIC ROLE OF AGE-RELATED IMMUNE DYSFUNCTIONS. (Ger.) Hijmans, W. (Inst. for Experimental Gerontology, Organization for Health Res. TNO 151, Lange Kleiweg, Rijswijk (ZH), The Netherlands). *Verh Dtsch Ges Pathol* (59): 118-127; 1975.

A literature review of the normal immune system, the age-dependent immunological deficit, and immunological aging is presented. Topics discussed include hormonal and cellular immunity, antibody diversity, Jerne's network model, immune deficiencies, autoimmunity, the limited heterogeneity of immunoglobulins, infection, autoimmune diseases, amyloidosis, and tumor formation. (22 references)

4237 CELL-MEDIATED IMMUNITY IN MURINE VIRUS TUMOR SYSTEMS. (Eng.) Herberman, R. B.; Kirchner, H.; Holden, H. T.; Glaser, M.; Haskill, S.; Bonnard, G. D. *In: Tumor Virus Infections and Immunity.* Crowell, R. L.; Friedman, H.; Prier, J. E., eds. (Baltimore: Univ. Park Press): pp. 147-164; 1976. 1976.

The current knowledge of cell-mediated immunity in murine virus tumor systems is reviewed. In the murine sarcoma virus (MSV) system, cell-mediated immunity results in the regression of tumors following a short period of tumor development after sc inoculation of tumor cells, but progressive tumor growth is possible in very young or immunosuppressed animals. Specific cell-mediated cytotoxicity reaches a peak at approximately 14 days, then rapidly declines. At 14 days after MSV inoculation, spleen cells and peritoneal exudate cells inhibit the in vitro proliferation of Rauscher RBL-5 cells. Populations of cells involved in these responses are discussed. Characteristics of the cell-mediated immune response to a Gross virus-induced tumor in rats are generally similar to those of the MSV system. Evidence for the presence of suppressor cells having the characteristics of macrophages is reviewed. These cells not only suppress lymphocyte proliferative responses, but they may be identical with the effector cells in the growth inhibition assay. Serum-mediated inhibition of cell-mediated immunity has frequently been observed with rat and mouse sera in the cytotoxicity assay, and serum factors from tumor-bearing rats also inhibit lymphocyte stimulation. Immunosuppression may also occur by the action of the tumor itself or by products of the tumor, as has been revealed in studies with the Gross virus-induced lymphoma (C58NT)D and the benzpyrene-induced lymphoma, EL-4. Caution is urged in translating in vitro data to in vivo systems in this complicated area. (29 references)

4238 TUMOR-SPECIFIC IMMUNITY. (Eng.) Smith, R. T. (Dept. Pathology, Univ. Florida, Gainesville, FL). *Adv Pathobiol* 4: 74-85; 1976.

The current knowledge of immunologic relationships between growing tumor and host is reviewed with reference to studies of both autochthonous human tumors and animal systems in which the syngeneic relationship between tumor and host limits the antigenic differences to those specific to the tumor. Most tumors present multiple antigenic and immunogenic determinants that are derived from the cell membranes. These antigens stimulate a polyclonal hyperimmune state in the host involving most or all elements of the immune response. Five elements of the tumor-host relationship are explored: the source and character of tumor antigens; the fate and distribution of these antigens; the components of the immune response that these tumor determinants evoke, as determined by both in vivo and in vitro analysis; the problems of blocking and application of in vitro analysis to the in vivo tumor-host relationship; and a tentative synthesis suggesting a balanced relationship between growing tumor and host immune systems. (14 references)

4239 HUMORAL FACTORS ABROGATING CELL-MEDIATED IMMUNITY IN THE TUMOR-BEARING HOST (Eng.) Baldwin, R. W. (Cancer Res. Campaign Labs., Univ. Nottingham, England); Robins, R. A. *Curr Top Microbiol Immunol* 72: 21-53; 1976.

A review of humoral factors that may interfere with cell-mediated immunity and serum factors that may correlate with tumor growth is presented in an attempt to understand why tumor-directed immunity fails in the tumor-bearing individual. Procedures designed to correct such defects in the tumor-bearing patient may be used in immunotherapy. In vitro studies have shown that circulating tumor antigen or immune complexes may play an important role in the tumor-host relationship by inhibiting the cell-mediated arm of the immune response. Blocking of target tumor cells by tumor-specific antibody or immune complexes may be important, since in vivo localization of immunoglobulin in tumor tissue has been reported. Studies on neuroblastoma patients indicate that complexes are more efficient than antibody alone in blocking tumor cells from attack by sensitized lymphoid cells. Tumor membrane antigen, released as a consequence of cell membrane synthesis and regeneration, may interact directly with sensitized lymphoid cells. Inhibition of T cell reactivity by interaction with the tumor-associated antigen is probably due to the direct blocking of effector cell receptors; this would account for the inhibition of short-term cytotoxicity detected by isotope release methods. Both in vitro and in vivo studies suggest that tumor-specific immune responses occur concurrently with tumor growth, but tumor immunity may be suppressed by tumors that grow beyond a certain size. Therapeutic protocols designed to eliminate putative blocking and inhibitory factors either from the circulation or microenvironment of the tumor may be devised from experimental animal studies. (131 references)

4240 CELL MEMBRANE MARKERS OF T AND B LYMPHOCYTES AND MONOCYTES IN LEUKEMIAS AND HEMATOSARCOMAS. (Eng.) Belpomme, D. (Institut de Cancerologie et d'Immunogenetique, Villejuif, France); Dantchev, D.; Joseph, R.; Santoro, A.; Lelarge, N.; Feuilhade de Chauvin, F.; Grandjon, D.; Mathe, G. *Adv Tumour Prev Defect Charact* 3: 357-366; 1976.

Identification of the membrane markers on human mononuclear cells allows these cells to be classified as either T lymphocytes, B lymphocytes, or monocytes. These classification techniques are applied to lymphomonocytoid hematological disorders. The use of membrane markers helps in diagnosing and classifying leukemias and hematosarcomas as well as increasing our understanding of the physiopathology of these diseases. However, only standardized methods should be used, and the results must be interpreted with care. The problems encountered with the marker methods available are summarized; and the importance of using a battery of tests, rather than any single one, to classify hematological disorders is stressed. The results of the various tests are summarized for: normal mononuclear cells, primary acute lymphoid leukemias, chronic lymphoid leukemias, hairy cell leukemias, leukemic lymphosarcomas, nonleukemic lymphomas, and Hodgkin's disease. (41 references)

4241 HODGKIN'S DISEASE. (Eng.) Greenwald, P.; Vianna, N. J.; Davies, J. N. P. *In: Cancer Epidemiology and Prevention. Current Concepts*. Schottenfeld, D., ed. (Springfield, IL: Charles C. Thomas): pp. 351-359; 1975.

Difficulties in the diagnosis of lymph node and splenic enlargement as Hodgkin's disease necessitate a microscopic examination to confirm the diagnosis. For a positive diagnosis, Reed-Sternberg cells must be seen in an appropriate histological background of lymphocytic and histiocytic proliferation. Both lymph nodal and extranodal tissue can be affected. The disease has been classified at different times as either an infectious disease or a malignant neoplasm; since 1948 it has been listed under neoplasms. Four subtypes of the disease are used in the most widely recognized classification system; they are useful as indicators of prognosis, but their relevance to etiology has not been demonstrated. The disease occurs all over the world, but both incidence and distribution vary according to unknown factors. Three subgroups of cases by age have been described: ages 0 through 14 yr, affecting men 85% of the time, declining in incidence in the US but high in South America, tropical Africa, and the Middle East; ages 15 to 34 yr, 54% male, increasing slightly in the US; and ages 50 and above, 63% male, decreasing in incidence. Questions as to whether the disease is one or two distinct entities are noted. Also, differences in incidence and mortality in relation to socioeconomic stratum and comparison with other diseases, especially infectious diseases of children, suggest that the cause may be an infectious or other environmental factor acting on persons of variable susceptibility. The rise in incidence rates in later childhood in the US may be related to the coincidental lymphoid tissue involution, since tonsillectomy seems to have a similar effect. Studies were made in New York State of the communicable nature of the disease using a group of high school students, their friends, and relatives. The direction of spread was primarily young to young or young to old. Herpesvirus appears to be a possible etiological agent. (35 references)

4242 SUBACUTE AND CHRONIC MYELOMONOCYTIC LEUKAEMIA: A DISTINCT HAEMATOLOGICAL ENTITY. (Eng.) Zittoun, R. (Service d'Hematologie de L'Hotel-Dieu, Place du Parvis-Notre-Dame, 75181 Paris Cedex 04, France). *Br J Haematol* 32(1): 1-7; 1976.

Subacute and chronic myelomonocytic leukemia (MML) are characterized. The mean age is about 65 and cases below age 50 are infrequent; the male: female ratio is about 1.2:1. The most common presenting symptom is anemia, but anemia was absent in about 10% of the patients in one series. Diagnosis is based on the finding of blood monocytosis, associated with signs of bone marrow proliferation and/or myeloid dysplasia. Total WBC counts below 2,000/μl or above 50,000/μl are rare. Monocyte morphology is often abnormal. One constant and characteristic finding is increased serum lysozyme. The main diagnostic difficulty is differentiation from carcinoma with or without bone marrow metastases; the necessity of ruling out other monocytotic conditions is stressed. Other major sources of diagnostic difficulty are refractory anemia with an excess of myeloblasts and acute MML. Chronic MML could be viewed as a spontaneous adaptation by a leukemic patient to his disease. This may be a future area of research in the treatment of leukemia--attempting to produce increased maturation of leukemic stem cells. In some cases of acute MML, chemotherapy has produced complete remission with persisting hematological abnormalities like those observed in chronic MML. (17 references)

4243 OCCURRENCES OF CENTRAL NERVOUS SYSTEM TUMORS, WITH SPECIAL REFERENCE TO RELATIVE GENETIC FACTORS. (Eng.) Mulcahy, G. M.; Harlan, W. L. *In: Cancer Genetics*. Edited by H. T. Lynch. (Springfield, IL: Charles C. Thomas): pp. 263-325; 1976.

The significance of the genetic factor in relation to the etiology and pathogenesis of CNS tumors (both human and experimental) is reviewed. Malignant CNS tumors are emphasized, but benign intracranial and intraspinal tumors such as meningioma, are also discussed; in addition, neuroblastoma, which may occur in the brain, is included. There are many relationships between genetic factors and CNS tumors, both in humans and experimental animals. Von Recklinghausen's disease and tuberous sclerosis are examples of human disease associated with CNS disorders. Several (but not all) family surveys have indicated that certain families have an increased incidence of CNS tumors. CNS tumors often occur in the apparent absence of any specific genetic influence, although general genetic susceptibility must be assumed. It is suggested that a large-scale prospective study of brain tumor patients, their relatives, and suitable controls should be undertaken. (660 references)

4244 INTRACRANIAL CHORDOMA. (Eng.) Sassin, J. F. *In: Handbook of Clinical Neurology. Part III: Tumours of the Brain and Skull*. Edited by P. J. Vinken, G. W. Bruyn. (Amsterdam: North-Holland Publishing Co.): vol. 18: pp. 151-164; 1975.

Although intracranial chordoma is relatively uncommon, it is difficult to diagnose because of its inaccessible location at the base of the skull. Chordomas are derived from remnants of the fetal notochord. The location of aberrant notochord tissue correlates well with the location of chordomas in later life. The incidence of intracranial chordoma ranges from 0.15% to 0.2%, according to various studies. Chordoma does not manifest itself clinically until middle life, with a peak incidence in the fourth and fifth decades. Death usually occurs 3-4 yr after diagnosis, and men are affected more frequently than women. Headache is the single most common symptom. Nasal stuffiness or obstruction, pain, and purulent or bloody nasal discharge are also common. The hallmark of neurological involvement in chordoma is multiple cranial neuropathies, which are generally insidious and often slowly progressive. They are usually unilateral. The symptoms for each location of chordoma are given. Plain radiographs of the skull in multiple projections are the most helpful diagnostic aids, and neuroradiologic contrast studies can confirm the presence of a mass lesion. Arteriographic and pneumoencephalographic signs of elevation may be seen in cases of lateral expansion. Biopsy of the nasopharyngeal portions of the tumor are also helpful. Death is usually the direct result of nervous system involvement. A 5-yr survival free of disease is estimated to be under 10%. (30 references)

4245 LUNG CANCER: A REVIEW. (Eng.) Adler, R. H. (No affiliation given). *West Indian Med J* 24(4): 210-217; 1975.

The etiology, pathology, diagnosis, and treatment of lung cancer are reviewed. The major etiological factor in lung cancer appears to be atmospheric air pollution, with cigarette smoking probably being the greatest pollutant. Bronchogenic carcinoma encompasses several morphologically different entities: epidermoid carcinoma; adenocarcinoma, including bronchioloalveolar carcinoma; large cell carcinoma;

small cell (anaplastic) carcinoma; and bronchial adenoma. Cough is the most common presenting symptom, and hemoptysis is present in over half of all lung cancer patients. Other symptoms include hoarseness, dyspnea, cardiac dysfunction, dysphagia, superior vena caval obstruction, pancoast or superior sulcus syndrome, chest pain, and symptoms and signs of pneumonitis that fail to clear up with appropriate therapy. Diagnostic procedures include cytological studies of sputum, bronchoscopy with brushing and biopsy, bronchography, lymph node biopsy (scalene lymph node biopsy, mediastinoscopy, mediastinotomy), needle biopsy, and liver biopsy. Surgery remains the treatment of choice. When tumor and nodes can be adequately excised, lobectomy is preferable to pneumonectomy. Radiotherapy is used primarily for palliation, but it is indicated over surgery for small cell carcinoma. Adjuvant chemotherapy for lung cancer has been disappointing, but palliation has been obtained with primary chemotherapy. Immunotherapy might prove valuable in that the lung cancer patient appears to have an altered immune system in association with his tumor. (18 references)

4246 GASTRIC CARCINOMA. (Ger.) Mitschke, H. (Pathologisches Institut, Universitaet Hamburg, 2 Hamburg 20, Martinistrasse 52, West Germany). *Med Monatsschr* 29(8): 347-355; 1975.

The pathogenesis, precarcinomatous state, and patterns of early carcinoma are reviewed. Two potentially pathological processes alter normal gastric mucosa with increasing age. In chronic gastritis the regeneration zone is displaced superficially, so that DNA-synthesizing cells appear on the surface epithelium. In the resting phase of intestinal metaplasia, the proliferation zone is displaced to the glandular level. Polypoid tumors and differentiated tubular structures typical of adenocarcinoma, respectively, are the terminal expressions of each process. The increasing, age-linked vulnerability of the mucosa is further conditioned by diminished cell migration and contact and by disturbances in differentiation, which lead to early ulcerative erosion. Chemically induced gastric carcinoma with a nitrosoguanidine compound exhibits these processes in animal models. The mucosa becomes atrophied, with superficial erosions. After a regenerative phase, a carcinoma develops, with mucosal hyperplasia around the eroded area and adenomatous hyperplasia. (75 references)

4247 THE ETIOLOGY OF COLON CANCER. (Eng.) Hill, M. J. (Central Public Health Lab., London, England). *CRC Crit Rev Toxicol* 4(1): 31-82; 1975.

Studies of the incidence of colon cancer in populations living in various parts of the world, taken in conjunction with studies of migrant populations, indicate that genetic factors do not play a major part in the etiology of colon cancer. The factors of overwhelming importance are environmental, especially diet (meat and dietary fat). Associated diseases (such as atherosclerotic heart disease) and associated cancers (such as prostate, breast, and corpus uteri) indicate a role for steroids in the etiology. The most likely carcinogen or cocarcinogen is an endogenous steroid, in particular the bile acids. Two bile acids, deoxycholic acid and lithocholic acid, have been shown to be cocarcinogenic in the large bowel. There are two major theories put forward to explain the etiology of colon cancer. Both involve the gut bacteria and both postulate that a carcinogen or cocarcinogen is produced by bacteria from bile acids. The fiber theory attempts to relate the incidence of the disease to the amount of dietary fiber by suggesting that the transit time of material through the gut is of paramount importance. The fat theory suggests that the concentration of bile acids in the large intestine is the most important factor. The weight of evidence to date is strongly in favor of the fat theory. (304 references)

4248 THE PRECURSOR TISSUE OF ORDINARY LARGE BOWEL CANCER. (Eng.) Lane, N. (Columbia Univ. Coll. Physicians Surgeons, New York, NY 10032). *Cancer Res* 36(7): 2669-2672; 1976.

The evolution of large bowel carcinoma via its precursor tissue was traced by morphological studies. Hyperplastic polyps are 10 times as common as adenomas and must be distinguished from them, since they are unrelated as a precursor tissue to either adenomas or carcinomas. Only adenomas are relevant to the development of the common moderately and well-differentiated large bowel cancer. Depending on three related factors (increasing size, a sessile rather than pedunculated mode of growth, and a villous rather than tubular microscopic architecture), minute (1 to 2 mm) or microcancer may be found with increasing frequency in adenomas. However, minute or microcancer has not been observed in normal mucosa; that is, mucosa unassociated with adenomatous tissue. The same findings obtain in familial polyposis, in which adenomas (but not carcinomas) as small as one or two crypts have been found in grossly normal areas of mucosa. Direct one-step transformation from normal crypt cells to cancer, without formation of adenomatous epithelium, does not seem to be the usual pathway. (11 references)

4249 CURRENT MORPHOLOGICAL PROBLEMS OF BREAST CARCINOMA. (Ger.) Hort, W. (Pathologisches Institut der Universitaet, Robert-Koch-Str. 5, 3550 Marburg/Lahn, West Germany); Kalbfleisch, H. *Therapiewoche* 26(6): 767-773; 1976.

A review of mastopathy, lobular carcinoma in situ, intraductal carcinoma, and intramammary calcification is presented from a histogenetic perspective. Mastopathies, which comprise 75% of all benign mammary masses and occur most frequently in the fourth or fifth decade of life, are thought to have an endocrine etiology. They are most often fibrocystic, with connective tissue proliferation and dilation of ducts of acini. However, the incidence of breast carcinoma is somewhat higher in women with mastopathies than in those without mastopathies. Accordingly, three grades of mastopathy can be distinguished: without proliferation or atypia, with proliferation and without atypia, and with both; the cancer rises through these levels from 0% to 40%-50%.

Atypias are most conspicuously characterized by a shift in the nuclear: cytoplasmic ratio. Each mammary carcinoma begins within the epithelium, duct, ductule, or lobule. The miniscule dimensions of an incipient lobular tumor are clinically hidden; eventually the acini enlarge, the basal membrane is compromised, and infiltration begins. Intraductal carcinomas, which frequently develop into the comedo type, are also clinically inert, even after the onset of invasion. The presence of microcalcification does not indicate tumor formation. (26 references)

4250 INFLAMMATION AND INFESTATION OF THE TESTIS AND PARATESTICULAR STRUCTURES. (Eng.) Morgan, A. D. *In: Pathology of the Testis*. Edited by R. C. B. Pugh. (Oxford: Blackwell Scientific Publications): pp. 79-138; 1976.

The inflammatory conditions of the testis, epididymis, spermatic cord, tunica vaginalis, and scrotum due to bacteria, fungi, viruses, protozoa, and parasites as well as lesions of doubtful or unknown etiology are reviewed. Neoplasia is considered in several of the differential diagnoses. (158 references)

4251 MULTIPLE ENDOCRINE NEOPLASIA TYPE II. (Eng.) Wells, S. A., Jr. (Duke Univ. Medical Center, Durham, NC 27710); Ontjes, D. A. *Annu Rev Med* 27: 263-268; 1976.

The clinical characteristics, methods of diagnosis, and treatment of multiple endocrine neoplasia type II (MEN-II) are described. The syndrome is associated with medullary carcinoma of the thyroid gland (MCT), pheochromocytoma(s), and parathyroid hyperplasia. MCT is usually diagnosed first. Diagnosis is based on the production of thyrocalcitonin by the MCT, which is greatly facilitated by stimulation with pentagastrin. Screening patients for the presence of pheochromocytoma(s) should consist of collecting 24-hr urine samples for epinephrine, norepinephrine, metanephrine, normetanephrine, or vanillylmandelic acid. Parathyroid hyperplasia occurs in about 60% of patients with MEN-II, and this is characterized by grossly enlarged parathyroid glands and hypercalcemia. The treatment of MEN-II is primarily surgical, and MCT is best treated by at least total thyroidectomy. The reported 10-yr survival for MEN-II is 60%-70% and depends on the presence of lymph node involvement at the time of the initial tumor resection. Attention in the future should be focused on diagnosing MCT at the earliest possible time in MEN-II family members.

4252 PARAENDOCRINE SYNDROMES IN THE PATHOGENESIS OF MALIGNANT GROWTH. (Rus.) Kashulina, A. P. (P. A. Gertsen Moscow Inst. Oncology, Moscow, USSR); Tereshchenko, I. P. *Patol Fiziol Eksp Ter* (6): 79-85; 1975.

Paraneoplastic syndromes, a term commonly used for various clinical complexes appearing as a result of the action of specific and nonspecific products of tumor metabolism (disappearing upon tumor removal and reappearing during relapse and metastasis), can be subdivided into neurotropic, autoimmune, and endocrine disturbances. Among the latter (a result of ectopic production of hormones or hormonelike substances with cancerous tissue) are cases in which tumors causing paraneoplastic endocrine syndromes (PNES) emerge from endocrine organs but secrete uncharacteristic hormones. The capacity of malignantly regenerated cells for ectopic production of hormones has been an area of intensive research. In certain cases, the same PNES can be caused by different biologically active substances. The biochemical heterogeneity of hypercalcemia and hypoglycemia attests to the variety of pathogenetic mechanisms of PNES and emphasizes the need for a differentiated approach to them when therapeutic measures are undertaken. Clinical manifestations of hormonal hyperproduction in a cancerous organism and the presence of hormonelike substances in the blood and cancerous tissue are closely connected. The recognition of PNES and hormonelike substances may help diagnosis and treatment of malignant diseases. (105 references)

4253 RARE MELANOCYTIC AND NEVOCYTIC NEVI. (Ger.) Frenk, E. (Centre hospitalier universitaire vaudois, CH-1011 Lausanne, Switzerland); Mevorah, B.; Delacretaz, J. *Hautarzt* 26(12): 619-624; 1975.

The frequency, clinical picture, histopathology, differential diagnosis, development, and management of various rare cutaneous melanocytic and nevocytic nevi are discussed. Nevus spilus, benign juvenile melanoma (spindle cell and epitheloid cell nevus), blue nevus, mongolian spot, and nevus Ota are included. Variants of benign juvenile melanoma are known to change into intracutaneous nevi, with spontaneous involution, lifetime persistence, and possible transformation to malignancy. A precancerous state is sometimes attributed to blue nevus and mongolian spot. Frank cutaneous melanomas have been based on nevus Ota; the brain and the eye are other sites of tumor localization. (54 references)

4254 PATHOLOGY OF MALIGNANT MELANOMA--PART 1. (Eng.) Woodruff, J. M. (Memorial Sloan-Kettering Cancer Center, 1275 York Ave., New York, NY 10021). *Clin Bull* 6(1): 15-23; 1976.

The pathology of malignant melanomas arising at different anatomical sites is reviewed. In Caucasians, the majority originate from melanocytes situated in the basal layer of the epidermis. The melanomas can arise from junctional and compound nevi, non-nevalbearing skin, lentigo maligna, and xeroderma pigmentosum. Melanocytes deposited in the dermis give rise to intradermal nevus, giant pigmented nevus, nevus of Ota, and blue nevus. Tumors arising from the squamous mucous membranes of the oral mucosa, oropharynx, larynx, esophagus, vagina, cervix, anus, and vulva are generally highly malignant. Malignant melanomas of the respiratory mucosa arise almost exclusively in the upper respiratory tract, while melanomas of the eye arise in the uveal tract and affect the choroid, ciliary body, and iris, in decreasing order of frequency. Prognosis in uveal melanomas is based on histologic type. Primary meningeal melanoma is an uncommon tumor that presumably arises from melanoblasts normally present in the pia of the spinal cord and ventral aspect of the brain. Those of the salivary and adrenal glands, gallbladder, and ovary are more often metastatic than primary. Malignant melanoma may arise from neval cell nests in lymph nodes. Primary malignant melanoma cells of the skin are usually polygonal and bear prominent, vesicular, or hyperchromatic, bizarre nuclei. Dermal invasion is usually conspicuous. The tumor often consists of nests or sheets of epithelioid and/or spindle cells, although some epithelioid melanomas have pleomorphic giant cell features and some are partially or exclusively nevoid in makeup. Frequent associated findings are acanthosis or pseudoepitheliomatous hyperplasia of the epidermis and surface ulceration. (45 references)

4255 THE ETIOLOGY AND EPIDEMIOLOGY OF BREAST CANCER. (Ger.) Dallenbach, F. D. (Deutsches Krebsforschungszentrum, 69 Heidelberg 1, Berliner Strasse 29, West Germany). *Fortschr Med* 93(28): 1323-1325; 1975.

A brief review summarizes current data on mammary cancer incidence in mammals, the effects of early pregnancy, endocrine effects, viral genesis, and geographic and congenital factors. Breast cancer in most mammals, except those under domestication, is infrequent. The cow, whose udder is exposed to frequent trauma and inflammation, is nearly entirely free. Cats and dogs, who are a part of the human ambience, have relatively high frequencies. The fact that a successful teenage pregnancy, but not a first pregnancy at later ages, tends to confer resistance to breast cancer in later life receives a novel explanation: carcinogenic xenobiotics, stored by estrogen-stimulated, developing mammary epithelium at puberty, are expelled during teenage parturition (when fat metabolism is elevated a hundredfold and cell division is active), before cell transformation can occur. This self-purification cannot occur at later ages when epithelial cells are already transformed and the response options to estrogen stimulation are restricted to cell division and there is no longer cell differentiation. Thus, the reason for negligible bovine mammary cancer is simply that the cow is constantly lactating and, so, self-purifying. (4 references)

4256 THE SEARCH FOR WOMEN AT HIGH RISK. (Eng.) Leis, H. P., Jr.; Raciti, A. *In: Risk Factors in Breast Cancer.* Edited by B. A. Stoll. (Chicago, William Heinemann Medical Books): New Aspects of Breast Cancer, vol. 2, pp. 207-225; 1976.

Both experimental and clinical observations indicate that the development of breast cancer is not a chance event that occurs randomly throughout the population. Certain women are in a high risk group. The major predictors of high risk include sex, age, familial history of breast cancer, parity, and previous cancer in one breast, and the precancerous type of fibrocystic disease. Over 99% of the cases occur in women, 75% in those over 40 yr. The hereditary forms are more frequent than is generally suspected. They have an early age at onset, tend to be bilateral or multiple, and seem to fit the two-step mutational concept. The risk of breast cancer in women with one or two children is 1.5 to 2.0 times that of women with three or more children. Although parity before the age of 25 exerts a definite protective influence, pregnancy after 35 may well carry a risk greater than that for nulliparous women. The risk of developing cancer in the second breast after the first has been removed is about five times greater than the risk of initial breast cancer in the general population. The presence of fibrocystic disease of the breast also increases the risk; in fact, there may be a common hormonal predisposition for both diseases. Prominent predictors of high risk include viral factors, immunological factors, genetic factors, radiation exposure, cancer in other organs, alteration in the hormonal milieu (including estrogen), castration and menstrual activity, and etiocholanolone and/or androsterone excretions. Possible predictors of high risk of breast cancer have varied statistical significance. They include high dietary fat, chronic psychogenic stress, hormone administration, trauma, chemical agents, hypothyroidism, living in the Western Hemisphere, upper socioeconomic status, living in a cold climate, being of the white race, and being Jewish. (103 references)

4257 THE SO-CALLED STROMATOSIS--ANGIOBLASTOMATOSIS OR A FORM OF ENDOMETRIOSIS? (Ger.) Bonk, U (Pathologisches Institut Nymphenburg, 8 Munich 50, Dachauer Strasse 188, West Germany); Gedeon, W.; Occhionero, F. *Med Klin* 70(24): 1031-1038; 1975.

Stromal endometriosis (SE) is discussed in terms of nomenclature, incidence, patient age, clinical picture, surgical findings, histopathology, prognosis, and pathogenesis. Four theories explaining the stromal islands in the myometrium are reviewed critically, as follows: SE as a form of adenomyosis or endometriosis, SE as a variant of mucosal sarcoma, SE as a hemangiopericytoma, and SE as a uterine angioblastomatosis. Deficiencies in each explanation are exposed. The intimate vascular relation of the tumor cells is seen as the central pathological expression, whose etiology could be explained by angioblastomatotic differentiation of primitive mesenchymal cells. (40 references)

4258 AETIOLOGY AND EPIDEMIOLOGY. (Eng.) Miller, R. W. *In: Cancer in Children. Clinical Management.* Edited by H. J. G. Bloom; J. Lemerle; M. K. Neidhardt; P. A. Voute. (New York: Springer Verlag): pp. 3-8; 1975.

The role of epidemiology in implicating environmental and genetic factors in carcinogenesis is reviewed. Transplacental chemical carcinogenesis was detected by an epidemiologic study following the development of adenocarcinoma of the vagina in young women whose mothers had taken diethylstilbestrol during pregnancy. A registry has been created for these mothers, and their children constitute a cohort that can be examined periodically to detect tumors early and to determine adverse health effects in relation to drug dosage, latency, and interval of susceptibility during gestation. Persons at high risk for leukemia have in common a distinctive genetic feature, aneuploidy in Down's syndrome, chromosomal breakage after exposure to ionizing radiation or benzene, and chromosomal fragility in Bloom's syndrome or Fanconi's aplastic anemia. Another group of diseases links Wilms' tumor, adrenocortical neoplasia, and primary liver cancer with each other and with other manifestations of growth excess. Other malformations with high risk of cancer include D-deletion syndrome (retinoblastoma) and dysgenetic ovaries (gonadoblastoma). Hereditary neoplasms have a high penetrance, with 80%-90% of persons with the deleterious gene developing cancer. The concordance rate is virtually 100% when one of a pair of monozygotic twins develops leukemia before the first birthday. Hamartomatous syndromes, genetically transmitted, could account for the clustering of cancers in families. Although the viral etiology of cancer has received great attention recently, epidemiologic methods have not so far implicated any virus in human carcinogenesis. (39 references)

4259 NURTURE AND NATURE IN CANCER OF THE BREAST AND THE ENDOMETRIUM. (Eng.) De Waard, F. *In: Host Environment Interactions in the Etiology of Cancer in Man. Proceedings of a Meeting Held at Primosten, Yugoslavia, 27 August-2 September 1972.* International Agency for Res. on Cancer (Lyon, France): Natl. Insts. Health (USA): pp. 121-130; 1973.

Correlations computed from data on cancer incidence in five continents among women (notably, breast cancer) were tabulated to see if there are any environmental effects on endocrine regulation. There were no correlations between cervical cancer and any of the other neoplasms, whereas colon cancer was highly correlated with breast and endometrial cancer and slightly less so with ovarian cancer. Climate or geographic latitude has no effect; it is suggested that the low incidence of these last four cancers in Japanese women may be due to nutrition. The age-specific breast cancer incidence curves of most populations in Asia, Africa, and the Middle East rose up to age 50, followed by a leveling-off or a decline; in Western countries the leveling-off phenomenon was followed by a steep rise after age 55. In the U.S., however, this phenomenon was not usually seen. The rising incidence of breast cancer does not seem to follow a simple relationship to age. An increase in potency of a carcinogenic stimulus may, with time, overcome the postmenopausal leveling-off trend seen most clearly in populations following a non-Western way of life. A hypothesis is presented to account for geographical differences in breast cancer incidence: (a) there may be two etiologically different types of breast cancer (with different age distributions that overlap in the 40- to 50-yr range). (b) The type that usually occurs in the premenopausal age group may be related to a hormonal abnormality involving ovarian function. (c) The second type, which occurs mostly in the postmenopausal group, may be etiologically related to nutritional habits prevalent in the Western world, especially to obesity. Results from a study of the relationship between urinary sediment smears in postmenopausal women and breast cancer are summarized; the smears classified as intermediate (rather than estrogenic or atrophic) are associated with the highest risk; overweight is also a risk factor. It is suggested that lean women in Western countries might have an incidence curve similar to that of Japanese women. It is recommended that future studies include data on the weight and height of all new breast cancer patients to determine how many of the geographical differences in breast cancer incidence can be explained by measurable population characteristics related to overweight. (17 references)

4260 GEOGRAPHIC, REPRODUCTIVE AND SOCIOBIOLOGICAL FACTORS. (Eng.) Thomas, D. B.; Lillenfeld, A. M. *In: Risk Factors in Breast Cancer.* Edited by B. A. Stoll. (Chicago, William Heinemann Medical Books): New Aspects of Breast Cancer, vol. 2, pp. 25-53; 1976.

Geographic, reproductive, and sociobiological factors are surveyed, along with possible interrelationships between these factors and their mechanisms of action. Death rates due to breast cancer vary markedly; they tend to be high in the developed countries with populations of predominantly European descent and low in less-developed countries, especially those with non-Caucasian populations. In the past three decades the mortality rates in most predominantly European populations, if high, have remained stable, and if low, have increased. Environmental factors appear to have a greater effect on postmenopausal women than premenopausal women. Evidence of environmental factors has been obtained through immigrant studies, which show that migrants tend to develop breast cancer at a rate more comparable to the country of adoption than the country of origin. Single women are at a higher risk than married women, and married women who have never been pregnant are at a higher risk than those with children. A striking relationship between risk of breast cancer and age at either natural or artificial menopause has been noted. No associations with other menstrual factors have been found consistently. The disease occurs more frequently in women of higher socioeconomic status and in women who are obese or who have rich fat diets. Reserpine and ionizing radiation have also been implicated. In male breast cancer, many of the same risk factors have been observed. (78 references)

4261 FAMILIAL AND GENETIC PREDISPOSITION. (Eng.) Anderson, D. E. *In: Risk Factors in Breast Cancer.* Edited by B. A. Stoll. (Chicago, William Heinemann Medical Books): New Aspects of Breast Cancer, vol. 2, pp. 3-24; 1976.

Although positive family history has long been accepted as doubling a woman's risk of breast cancer, there is still doubt as to its etiological significance. Retrospective statistical studies have shown an aggregation of cancer, generally of the same origin, in relatives of patients. They have indicated approximately a two- to threefold excess of breast cancer in mothers, sisters, and daughters of patients compared with women in general. This could be the result of a polygenic mechanism, involving the action of several genes, presumably with small effects and accounting for only a relatively small portion of the total variation in breast cancer. Familial patients with bilateral breast cancer had a noticeably earlier average age at diagnosis than unilateral familial patients or nonfamilial patients. Early age and bilaterality appeared to have genetic significance, because the risk was about twice as high in first-degree relatives of patients with premenopausal diagnoses than in relatives of patients with postmenopausal diagnoses. These and other results have suggested a genetic heterogeneity in familial breast cancer. Twin and pedigree studies have provided some data in which a genetic basis for breast cancer cannot be dismissed readily. With respect to geographical differences, dietary factors and their effect on hormone synthesis and metabolism are now considered prime suspects. (112 references)

4262 FACTORS AFFECTING GROWTH OF NORMAL AND MALIGNANT CELLS IN VITRO.

(Eng.) Lipkin, G. (New York Univ. Sch. Medicine, New York, NY 10016); Rosenberg, M.; Knecht, M. E. *Biochem Pharmacol* 25(12): 1333-1337; 1976.

Cell growth patterns in vitro reflect the interplay of numerous factors that modulate cell surface characteristics, and understanding these interactions may bring forth new approaches to therapeutic intervention and control of neoplastic growth processes. Functional repair of malignant cells may be an effective alternative to eradicating cells to control disease. Contact inhibition of growth is the in vitro property the loss of which is most closely correlated with malignancy in vivo. The density-dependent growth limitation can be removed by several exogenous factors; a variety of overgrowth factors elaborated by transformed or embryonic cells can overcome the density dependence of nontransformed cells. Fibroblast growth factor stimulates the division of human diploid fibroblasts, overcoming the contact-inhibited state. Factors that promote cell growth and the loss of density-dependent restrictions include cAMP, phosphodiesterase inhibitors, prostaglandin E_1 and prostaglandin E_2, and inhibitors of proteases and hyaluronidase. The selective deletion of a high molecular wt (250 K) external glycoprotein following malignant transformation of chick embryo, mouse, and hamster fibroblasts has been described previously. A line of contact-inhibited hamster melanocytes can produce a high molecular weight external protein capable of restoring to malignant melanocytes the capacity for contact inhibition of growth. This inhibition involves a G_1 arrest, as in the classical case of diploid fibroblasts. Proteolytic enzymes can trigger cell division in resting cells and are present in elevated levels in transformed cells. Reported cellular events following protease treatment include loss of 250-K glycoprotein, altered levels of cyclic nucleotides, increased lectin agglutinability, increased nutrient transport, phosphorylation of histone and nonhistone proteins, and cell division. The clinical application of these findings may lead to new therapeutic approaches against neoplasms. (100 references)

4263 INFLUENCE OF GEOMETRY ON CONTROL OF CELL GROWTH.

(Eng.) Folkman, J. (Children's Hosp. Medical Center, Boston, MA); Greenspan, H. P. *Biochim Biophys Acta* 417(3/4): 211-236; 1975.

Geometric effects on untransformed and transformed cell growth are reviewed. When diploid or untransformed cells are plated on coverslips of plastic or glass, they stick and flatten out and proliferate until confluence is reached; when the same cells are suspended, they fail to proliferate. The major difference between these cells is their shape: cells in suspension are rounded or spheroidal and cells attached to plastic are flat and spread out. Inhibition of growth in the spheroidal state is characteristic of the majority of normal fibroblasts and epithelial cells. The general rule that flatness permits proliferation in a normal cell while roundness inhibits it has at least two analogs in experimental animals: (a) tumor production by implantation of cells coated on glass beads; and (b) foreign body carcinogenesis. In both cases, a flat configuration is necessary for tumor development to occur. Transformed cells can proliferate equally well in the flat state or the spheroidal state. Two-dimensional growth of transformed cell populations is not self-limited; in three-dimensional populations (in suspension culture) growth is limited by the shape of the population itself. The most likely mechanism is based upon the limiting effects of diffusion gradients or nutrients, oxygen, and catabolites that build up across the surface. Tumor cells that are able to make tumor angiogenesis factor thereby induce new capillary blood vessels from the host. These vessels penetrate the tumor and permit further rapid growth, thus creating a mechanism by which successful tumors escape the growth restriction imposed upon three-dimensional cell populations by geometry. It is suggested that geometric control of growth can operate on two levels, cell shape and population shape. In each situation, the growth restriction imposed by geometry can be overcome only if the cell acquires or expresses some new property. (49 references)

4264 INFLUENCE OF GEOMETRY ON GROWTH OF NORMAL AND MALIGNANT CELLS.

(Eng.) Folkman, J. (Children's Hosp. Medical Center, Boston, MA). *Adv Pathobiol* 4: 12-28; 1976.

The controlling influence of cell geometry on the growth of malignant and normal mammalian cells is reviewed. Brown-Pearce tumor cells growing in the aqueous humor of the anterior chamber of the rabbit eye ceased growing when spheroidal tumors reached less than 1 mm diameter. When the tumors were moved contiguous to the iris, new vessels penetrated the tumor and rapid growth ensued. L-5178Y spheroids containing approximately 3 x 10^5 cells were exposed to a single pulse of ^{3}H-thymidine. At less than 0.1 mm diameter, almost all cells were labeled, but beyond a diameter of 0.3 mm there was usually a central necrotic area, a middle zone of live but unlabeled cells, and an outer zone of labeled cells with accompanying mitoses. When this same population of cells was cultured in a flat configuration, unlimited growth developed. Malignant tumors in vivo generally exist in one of four configurations: spheroidal without capillaries, spheroidal with capillaries, flat sheets or thin cords, and ascites or bone marrow. Most carcinomas begin as microscopic avascular nodules that progress to the vascularized tumor. Human tumors in sheets or cords or in ascites form are very rare and usually occur only in late stages of disease. Although it is the shape of the tumor cell population that regulates growth, with normal cells it is cell shape that is important in controlling in vitro growth. The effect of shape on the in vivo growth of normal cells is more difficult to ascertain. The growth limitation of three-dimensional cell populations can be understood partly in terms of diffusion gradients and the access of nutrient and catabolic molecules to crowded cell populations. There is no satisfactory explanation of geometric control over individual normal cells. Variations in surface tension over the cell membrane may have a role in this mechanism. (25 references)

4265 BIOCHEMICAL STUDIES ON CELL ASSOCIATION AND ITS RELEVANCE TO NEOPLASIA. (Eng.) Humphreys, T. *In: Cellular Membranes and Tumor Cell Behavior. Annual Symposium on Fundamental Cancer Research, 1975.* (Baltimore: Williams and Wilkins Co.): pp. 173-192; 1975.

Biochemical studies of cell aggregation-enhancing factors in marine sponges, chicken and mouse embryos, mouse teratoma cells, and slime mold cultures are reviewed. An aggregation factor was isolated from the tissues of many sponge species by dissociation in cold calcium and magnesium-free seawater. Experiments involving the aggregation of cells from two different species in a mixed suspension indicated that the reaction of factor with cell surface is species specific. The factor from one sponge species has been purified and characterized as a large, negatively charged proteoglycan complex of 20 million daltons. Electron microscopy indicated that the factor consisted of fibers arranged as a central circle with arms branching off. Aggregation studies with chick and mouse embryos suggest that a receptor in the cell surface binds a specific polysaccharide in the enhancing factor. The enhancing factor protein then projects from the cell to bind the ligator, which is at least divalent and able to bind two cells together. It is further suggested that the ligator is analogous to the sponge aggregation factor and may be a related kind of molecule. Aggregation of trypsin-dissociated mouse teratoma cells growing as an ascites tumor of embryoid bodies proceeded much more rapidly in the presence of the ascites fluid, suggesting that the fluid contains a factor that is actually the molecule being synthesized from the polysaccharide precursors (glutamine or glucosamine) on which aggregation is dependent. The factor appears to be a ligand; it is specific; and partial characterization indicates that it is a large molecule containing protein and polysaccharide. Factor molecules are much less tightly bound to the tumor cells and tend to be lost rapidly, which would explain the high concentration of aggregation factor in the ascites fluid. As the molecular systems for cellular association are elucidated, it should be possible to determine the molecular basis for the change in a cell that causes it to leave its normal associations and become malignant. (36 references)

4266 SOME BIOPHYSICAL ASPECTS OF CELL CONTACTS IN METASTASIS. (Eng.) Weiss, L. *In: Cellular Membranes and Tumor Cell Behavior. Twenty-Eighth Annual Symposium on Fundamental Cancer Research, 1975.* (Baltimore: Williams and Wilkins Co.): pp. 361-382; 1975.

Theoretical considerations and experimental results concerning the biophysical aspects of cell contacts in metastasis are reviewed. Potential energy and viscous barriers tend to prevent cells from making contact with each other. Contact between cells involves escape from the average electrostatic situation that tends to prevent contact. At distances of less than 2-3 nm the question of surface charge topography may be more important than charge density. Alignment phenomena operating over hundreds of nanometers may result in probes from one cell making contact with lower-than-average charge density regions of another. Possible mechanisms for the latter involve cellular exudates either guiding microvilli to preexisting low charge density regions and/or exudates from microvilli causing charge rearrangements in opposing cell surfaces that are energetically suitable for contact. Among metastatic phenomena the arrest of circulating tumor cells at the vascular endothelium has been shown to involve an exudation product of interaction (fibrin); contact interactions between malignant cells and macrophages or lymphocytes also involve other exuded products, including the lymphokines. It is hypothesized that net, negatively charged molecules diffuse from the microvillus to approach closely enough to the flatter surface of another cell so that the tangential component of the electrostatic forces acts to displace neighboring mobile negatively charged molecules. Computations indicate that even though an approaching macromolecule would tend to rest in electrostatic equilibrium with respect to the opposing charged surface molecules, it could then be attracted closer to the surface by attractive Van der Waals' forces and cause further electrostatic displacement of the responding surface molecules. (48 references)

4267 CELL DENSITY AND GROWTH CONTROL. (Fin.) Vaheri, A. (Helsingin yliopiston virusopin laitos, 00290 Helsinki 29, Finland). *Duodecim* 91(22): 1392-1400; 1975.

Malignant cells seem to have many of those surface properties that are only temporary in normal cells freed from growth control. Cancer cells, as well as mitotic and trypsinized cells, are known to agglutinize easily because of the effect of lectins, such as concanavalin A. When lectin is marked with fluorescein or ferritin molecules, it is possible to see in a fluorescence or electron microscope the patching of antigens on the cell surface. In normal undivided cells patching occurs very slowly or not at all. Increased proteolytic enzyme activity is known to be characteristic of malignant cells. They create a so-called serine-type protease, which can be recognized by its ability to change proenzyme plasminogen in serum into active protease plasmin. Malignant cells also produce surface antigen, as do normal cells, but it does not stay on the cell surface. This chain of events: creation of cell surface protease, detachment of the glycoprotein that is part of the cell support structure, then increased lateral motion of lectin receptors, could explain the release of mitotic, trypsinized, and malignant cells from growth control. (43 references)

4268 CANCER-RELATED ASPECTS OF REGENERATION RESEARCH: A REVIEW. (Eng.) Donaldson, D. J. (Univ. Tennessee, Center Health Sciences, Memphis, TN 38163); Mason, J. M. *Growth* 39(4): 475-496; 1975.

Studies on tissue regeneration in animals are reviewed, with emphasis on epimorphic regeneration in urodele amphibians. Some tumors such as lymphosarcomas and melanomas can originate and grow in regeneration-competent appendages. Evidence exists that the growth of both of these tumors can be modified to some extent by initiating regeneration of the appendages containing them. With respect to lymphosarcoma, some situations (*Xenopus* limbs) show no control, while others (*Bufo* tails) do. Melanomas appear to be less malignant when growing in new regenerates. Iris and mesodermal tissues in regeneration fields are quite resistant to carcinogens, and potentially lethal epithelial tumors spontaneously differentiate in the tail field. Epimorphic regeneration fields may suppress tumor induction and control existing tumors. This influence may be mediated by bioelectric fields, which are ubiquitous in nature and appear to control many cellular events. Disruption of these bioelectric fields suppresses epimorphic regeneration and can lead to cancer in mammals, while applied electric fields alter regenerative events and cause tumor regression. Since membrane changes in cancer cells are important in their abnormal behavior, it is essential that the similarities and differences of cancer cell and regeneration cell membranes be delineated. (81 references)

4269 TUMOUR CYTOGENETICS AND TISSUE CULTURE. (Eng.) Rigby, C. C. *In: Pathology of the Testis*. Edited by R. C. B. Pugh. (Oxford: Blackwell Scientific Publications): pp. 160-163; 1976.

The cytogenetics of testicular tumors is briefly discussed. The modal number of chromosomes in seminomas may range between 61 and 88, and the range in teratomas is 52-58. The determination of the karyotype and chromosomal number may sometimes assist in making the distinction between an anaplastic seminoma and an undifferentiated teratoma. Abnormalities of chromosomal structure, although commonly encountered, are not specific for or diagnostic of histological type. Methods used for the preparation of tumor cells for cytogenetic studies are briefly summarized. Mitotic activity can be increased by first culturing the tissue in vitro, but this entails a risk of selective growth of some elements so that the final culture does not accurately represent the in vivo tumor. No long-lived cultures of testicular tumors have yet been achieved, but considerable attention has been directed toward using experimental tumors in animals. (17 references)

4270 ROLE OF DNA REPAIR IN MUTATION AND CANCER PRODUCTION. (Eng.) Trosko, J. E.; Chang, C.-C. *In: Aging, Carcinogenesis, and Radiation Biology. The Role of Nucleic Acid Addition Reactions.* Smith, K. C., ed. (New York: Plenum Press): pp. 399-442; 1976.

Against a background of the genetic and environmental factors that predispose to the development of cancer, the nature and nurture of mutagenesis and carcinogenesis are discussed. It appears that since most carcinogens, if not all, react with DNA and since many of these altered DNA molecules are substrates of various types of DNA replication and repair enzymes, mutagenesis might be one important mode of carcinogenic initiation, the other being gene derepression. Several human syndromes have genetic predisposition to cancer, as well as to other specific syndrome abnormalities. Initiation of DNA damage and mechanisms of repair are discussed. Factors influencing DNA repair include developmental factors, aging, and environmental factors. Although the molecular mechanism for gene regulation in eukaryotic cells is not well-understood at present, there are probably several means by which structural gene expression is altered. A mutation in the regulator segment could result in the derepression or repression of a structural gene. Activation of integrated viral information or the insertion and activation of viral DNA might lead to altered structural gene activity. Or, nonrepair of DNA lesions in the regulatory region might modify structural gene expression. If somatic mutagenesis is the result of defective repair, caused in part by programmed developmental constraints on ''error-free'' repair, then cancer and aging might be the biological consequence. (213 references)

4271 GENE REGULATION: SELECTIVE CONTROL OF DNA HELIX OPENINGS. (Eng.) Frenster, J. H. *In: Aging, Carcinogenesis, and Radiation Biology. The Role of Nucleic Acid Addition Reactions.* Edited by K. C. Smith. (New York: Plenum Press): pp. 53-66; 1976.

DNA helices must undergo openings in the form of localized strand separations to permit the onset of RNA synthesis during gene transcription and the onset of DNA synthesis during gene replication. Several examples of these openings have recently been recognized in both bacterial and animal cells. Recent findings concerning the quaternary structure of chromatin suggest that superhelical coils of DNA helices may favor DNA helix openings in DNA segments from which covering histones have been displaced, similar to the effects of such superhelical twisting on DNA helix openings in covalently closed circular bacterial and viral DNA. In animal cells, histones are usually in close apposition with underlying DNA templates and therefore must be displaced from such templates before gene transcription can occur. Evidence for the repositioning of displaced histones during inactivation of the DNA template has also been found with a variety of animal systems. The molecular mechanisms mediating histone displacement and derepression are complex and usually involve a variety of acidic macromolecules found in association with DNA in derepressed euchromatin complexes. Derepressor RNA is capable of hybridizing to double-stranded DNA in 5 M urea, apparently binding to the noncoding strand of DNA at a particular gene locus, thereby forming a DNA helix opening and freeing the remaining coding strand of DNA for messenger RNA synthesis. A variety of DNA molecules can also interact with specific portions of the cellular genome, inducing DNA helix openings and increasing RNA synthesis at the particular gene locus. Oncogenic viral DNA undergoes single-stranded integration with the host genome, forming a DNA helix opening and allowing both messenger RNA synthesis on the remaining free host DNA strand and antimessenger RNA synthesis on the remaining free viral DNA strand. Among the compounds capable of mediating DNA helix openings are nuclear RNA, acidic nonhistone proteins, saline-soluble proteins, lipoproteins, and nonintegrated exogenous DNA. (67 references)

4272 PROGNOSIS OF THE EFFECTIVENESS OF NEW ANTINEOPLASTIC DRUGS. (Bul.) Popov, D. (Acad. Med., Oncological Center, Bulgaria). *Onkologia (Sofia)* 12(3): 123-126; 1975.

A review is presented of the existing literature on predicting the effectiveness of new antineoplastic drugs and the principles that should be considered: (a) programming the compound's properties and predicting the results of its effect; (b) developing an experimental model; (c) prognosis based on the results and conclusions of experiments, in order to find the best ways of determining the most effective antineoplastic drugs. (19 references)

4273 JUNCTIONAL MEMBRANES IN NORMAL AND NEOPLASTIC TISSUES. (Eng.) Gilula, N. B. *In: Cellular Membranes and Tumor Cell Behavior. Twenty-Eighth Annual Symposium on Fundamental Cancer Research, 1975.* (Baltimore: Williams and Wilkins Co.): pp. 219-237; 1975.

Three types of cell junctions (tight junction, gap junction and the desmosome) and their potential significance in certain aspects of neoplastic growth are reviewed. The tight junction, which is characteristically present between epithelial cells, has been implicated in providing a barrier function for the intercellular diffusion of large molecules between cells. The gap junction, which has been isolated as enriched subcellular fractions from rat and mouse liver, has been implicated as the low-resistance pathway between cells. Metabolic coupling appears to be coincident with the presence of a low-resistance pathway, so that it is possible to consider the gap junction as the structural pathway for both ionic and metabolic coupling between cells. Desmosomes are most abundant in tissues that undergo significant stress, such as stratified squamous epithelium and the myocardium, and are responsible for maintaining cell-to-cell adhesion in the presence of unusual mechanical stress. There have been several studies that indicate that cell junctions may significantly decrease in a number of neoplastic tissues (cervical carcinomas, carcinomas of the liver, skin, and mammary gland, and fibrosarcomas), but they do not completely disappear. A qualitative change in tight-junctional permeability was seen in a mouse mammary adenocarcinoma. In some neoplastic cell culture systems, gap junctions are present with no detectable quantitative or qualitative alterations. It is concluded that there is not adequate information to determine whether junctional membranes are generally altered in neoplastic tissues, although in a few instances cell junctions are significantly reduced in number. It is important to determine if the reduction in cell junctions leads to altered adhesivity and cellular growth or whether the junctions are reduced as a result of neoplasia. (67 references)

4274 COMPARATIVE EVALUATION OF PLASMA MEMBRANE ALTERATIONS ASSOCIATED WITH NEOPLASIA. (Eng.) Smets, L. A.; van Beek, W. P.; Collard, J. G.; Temmink, H.; van Gils, B.; Emmelot, P. *In: Cellular Membranes and Tumor Cell Behavior. Twenty-Eighth Annual Symposium on Fundamental Cancer Research, 1975.* (Baltimore: Williams and Wilkins Co.): pp. 269-287; 1975.

Research performed by the Netherlands Cancer Institute on cellular membranes and tumor cell behavior is reviewed. Modulations in concanavalin A (Con A) agglutination of transformed cells resulting from variables such as cell-cycle position and cell-to-cell contact were investigated. Cell-cycle modulation of agglutinability is concluded to be an endogenous process that occurs independently of contact with neighboring cells. Other experiments indicated that the agglutinability of various transformed cell lines is a function of cell density or tissue culture time. The role of surface morphology as a factor in the cell cycle- and cell contact-mediated changes in agglutinability is currently being investigated. The usefulness of plasma membrane glycoprotein changes as an expression of malignancy was investigated by comparison of several pairs of malignant and normal cells. It is concluded that this biochemical marker is a general expression, occurring independently of the cell morphology or the malignant determinant of the various cell lines. Studies of the elution profiles of surface glycoproteins from spontaneously transformed 3T3-F cells, from untransformed cells, and from synchronized transformed cells suggest the following: (a) transformed cells contain surface glycopeptides with increased sialic acid density that are responsible for the horizontal shifts in the elution profiles; and (b) the elution profiles of surface glycoproteins from transformed cells are unstable with regard to the relative amounts of the various glycopeptides. Both phenomena may reflect a fundamental defect in sialyltransferase mechanisms in neoplastic cells. (22 references)

4275 TRANSPORT OF SUGARS ACROSS THE TUMOR CELL MEMBRANE. (Eng.) Hatanaka, M. *In: Cellular Membranes and Tumor Cell Behavior. Twenty-Eighth Annual Symposium on Fundamental Cancer Research, 1975.* (Baltimore: Williams and Wilkins Co.): pp. 141-172; 1975.

Factors affecting sugar transport in general and evidence for alteration of the sugar transport system across the tumor cell membrane are reviewed. Sugar transport is affected by changes in the physiological state, including cell-cycle phase; differentiation; population density; and the environmental milieu. Uptake of D-glucose, D-glucosamine, and 2-deoxy-D-glucose is increased during the transformation of chicken cells by Rous sarcoma virus. Similar results have been seen in mammalian cells infected by mammalian sarcoma viruses. The uptake of other precursor molecules by mouse and chicken cells was also higher after sarcoma virus infection, but the magnitude of the sugar uptake induced by sarcoma virus was always overwhelming. The most easily detectable biochemical change during the early stage of transformation is altered transport of sugar, followed by enhanced hexokinase activity, and glycolysis near the completion of transformation. Dose-response studies have indicated a linear relationship between the rate of uptake of 2-deoxy-D-glucose by murine sarcoma virus-infected murine cells and the activity estimated by focus assay of the sarcoma virus. Studies using 3-O-methyl-D-glucose as the substrate indicated that the rate of sugar transport increased too rapidly to be measured accurately after cell transformation. The saturation levels of 3-O-methyl-D-glucose in the transformed cells were directly proportional to the external concentration, and the transport rate out of the tumor cells was slower than the rate of entry into the cells. It is concluded that 3-O-methyl-D-glucose is transported by the cells using a carrier-mediated facilitated diffusion system, but the precise kinetics remain to be studied. Manipulation of the sugar transport may prove useful for the control of certain malignant diseases. (74 references)

4276 CYCLOCHLOROTINE. (Eng.) IARC Working Group. *In: IARC Monographs on the Evaluation of the Carginogenic Risk of Chemicals to Man: Some Naturally Occurring Substances. Meeting Held in Lyon, 7-13 October 1975.* International Agency for Res. on Cancer. (Lyon, France): vol. 10, pp. 139-144; 1976. (13 references)

4277 GRISEOFULVIN. (Eng.) IARC Working Group. *In: IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Man: Some Naturally Occurring Substances. Meeting Held in Lyon, 7-13 October 1975.* International Agency for Res. on Cancer. (Lyon, France): vol. 10, pp. 153-161; 1976. (46 references)

4278 LUTEOSKYRIN. (Eng.) IARC Working Group. *In: IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Man: Some Naturally Occurring Substances. Meeting Held in Lyon, 7-13 October 1975.* International Agency for Res. on Cancer. (Lyon, France): vol. 10, pp. 163-169; 1976. (24 references)

4279 MONOCROTALINE. (Eng.) IARC Working Group *In: IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Man: Some Naturally Occurring Substances. Meeting Held in Lyon, 7-13 October 1975.* International Agency for Res. on Cancer. (Lyon, France): vol. 10, pp. 291-302; 1976. (48 references)

4280 PARASORBIC ACID. (Eng.) IARC Working Group. *In: IARC Monogrpahs on the Evaluation of the Carcinogenic Risk of Chemicals to Man: Some Naturally Occurring Substances. Meeting Held in Lyon, 7-13 October 1975.* International Agency for Res. on Cancer. (Lyon, France): vol. 10, pp. 199-204; 1976. (13 references)

4281 PATULIN. (Eng.) IARC Working Group. *In: IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Man: Some Naturally Occurring Substances. Meeting Held in Lyon, 7-13 October 1975.* International Agency for Res. on Cancer. (Lyon, France): vol. 10, pp. 205-210; 1976. (21 references)

4282 TUMOR CELL SURFACE AND METASTASIS: DYNAMIC CHANGES IN NEOPLASTIC MEMBRANE STRUCTURE AND THEIR RELATIONSHIP TO TUMOR SPREAD. (Eng.) Nicolson, G. L.; Robbins, J. C.; Winkelhake, J. L. *In: Cellular Membranes and Tumor Cell Behavior. A Collection of Papers Presented at the Twenty-Eighth Annual Symposium on Fundamental Cancer Research, 1975.* Univ. Texas System Cancer Center, Houston. (Baltimore, MD: The Williams and Wilkins Co.): pp. 81-127; 1975. (144 references)

4283 RETRORSINE. (Eng.) IARC Working Group *In: IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Man: Some Naturally Occurring Substances. Meeting Held in Lyon, 7-13 October 1975.* International Agency for Res. on Cancer. (Lyon, France): vol. 10, pp. 303-312; 1976. (31 references)

4284 SENECIPHYLLINE. (Eng.) IARC Working Group. *In: IARC Monographs on the Evaluation of* *the Carcinogenic Risk of Chemicals to Man: Some Naturally Occurring Substances. Meeting Held in Lyon, 7-13 October 1976.* International Agency for Res. on Cancer. (Lyon, France): vol. 10, pp. 319-325; 1976. (23 references)

4285 ANOTHER VIEW OF HERPES SIMPLEX VIRUS TYPE 2 (Letter to Editor). (Eng.) Rotkin, I. D. (Univ. Illinois Coll. Medicine, Chicago, IL). *JAMA* 235(20): 2188-2189; 1976. (5 references)

4286 VIRAL DISEASES OF THE FEMALE GENITAL TRACT. (Ger.) Klingmueller, G. (Univ.-Hautklinik, D-5300 Bonn-Venusberg, West Germany). *Gynaekologe* 8(3): 160-166; 1975. (53 references)

4287 TYPE C RNA VIRUSES: DIVERSE IMMUNOLOGICAL AND ETIOLOGICAL ROLES. (Eng.) Aoki, T. (Natl. Cancer Inst., Natl. Insts. Health, Bethesda, MD 20014); Chang, S.-S. *Adv Biosci* 12: 597-604; 1974. (18 references)

4288 HUMORAL HOST DEFENSE MECHANISMS AGAINST TUMORS. (Eng.) Ting, C.-C. (Natl. Cancer Inst., Bethesda, MD); Herberman, R. B. *Int Rev Exp Pathol* 15: 93-152; 1976. (480 references)

4289 HUMORAL IMMUNOSUPPRESSIVE FACTORS. (Eng.) Cooperband, S. R. (Boston Univ. Sch. Medicine, Boston, MA 02118); Nimberg, R.; Schmid, K.; Mannick, J. A. *Transplant Proc* 8(2): 225-242; 1976. (165 references)

4290 DISCUSSION. (Eng.) Roth, S. *In: Cell Surfaces and Malignancy. Meeting Held in Bethesda, September 11-13, 1974.* John E. Fogarty International Center for Advanced Study in Health Sciences and Natl. Insts. Health. (Bethesda, MD): pp. 191-193; 1975. (No references)

4291 PROBLEM OF RELATIONSHIPS BETWEEN IgA LEVEL, CANCER, AND ALCHOHOLISM (Letter to Editor). (Fre.) Thabaut, A. (Clinique des Maladies de l'Appareil Digestif, H.I.A. Beguin, 69, avenue de Paris, F 94160 Saint-Mande, France); Durosoir, J. L.; Laverdant, C. *Nouv Presse Med* 5(16): 1067-1068; 1976. (3 references)

4292 INTERNATIONAL VARIATION IN HIGH-RISK POPULATIONS. (Eng.) Muir, C. S. *In: Persons at High Risk of Cancer. An Approach to Cancer Etiology* Academic Press, Inc.): pp. 293-305; 1975. (32 references)

4293 SEXUAL FACTORS AND PREGNANCY. (Eng.) Henderson, B. E.; Gerkins, V. R.; Pike, M. C. *In: Persons at High Risk of Cancer. An Approach to Cancer Etiology and Control.* Fraumeni, J. F., Jr., ed. (New York: Academic Press, Inc.) pp. 267-284; 1975. (99 references)

4294 DIETARY FACTORS AND SPECIAL EPIDEMIOLOGICAL SITUATIONS OF LIVER CANCER IN THAILAND AND AFRICA. (Eng.) Wogan, G. N. (Dept. Nutrition and Food Science, Massachusetts Inst. Technology, Cambridge, MA 02139). *Cancer Res* 35(11/Part 2): 3499-3502; 1975. (10 references)

4295 DIETARY FACTORS AND CANCER IN ISRAEL. (Eng.) Modan, B. (Tel Aviv Univ. Medical Sch., Tel Aviv, Israel); Barell, V.; Lubin, F.; Modan, M. *Cancer Res* 35(11/Part 2): 3503-3506; 1975. (40 references)

4296 RELATIONSHIP OF PLUMMER-VINSON DISEASE TO CANCER OF THE UPPER ALIMENTARY TRACT IN SWEDEN. (Eng.) Larsson, L.-G. (University Hosp. Umea, 901 85 Umea, Sweden); Sandstrom, A.; Westling, P. *Cancer Res* 35(11/Part 2): 3308-3316; 1975. (37 references)

4297 CURRENT VIEWS ON BREAST CANCER (II). (Spa.) Estape, J. (No affiliation given). *Med Clin* 63(8): 441-444; 1974. (26 references)

4298 RELEVANCE OF ANIMAL STUDIES TO HUMAN DISEASE. (Eng.) Hegsted, D. M. (Harvard Sch. Public Health, Boston, MA 02115). *Cancer Res* 35(11/Part 2): 3537-3539; 1975. (8 references)

4299 MUTAGENESIS BY 9,10-ANTHRAQUINONE DERIVATIVES AND RELATED COMPOUNDS IN *SALMONELLA TYPHIMURIUM*. (Eng.) Brown, J. P. (Dynapol, 1454 Page Mill Road, Palo Alto, CA 94304); Brown, R. J. *Mutat Res* 40(3): 203-224; 1976.

Ninety 9,10-anthraquinone (AQ) derivatives and related anthracene derivatives were screened for mutagenicity with five *Salmonella typhimurium* strains with and without mammalian microsomal activation. About 35% of the compounds tested were considered to be mutagenic. Three patterns of mutagenesis were apparent. The first involved direct frameshift mutagenesis by certain AQ compounds bearing free hydroxyl groups. The most potent were anthragallol (1,2,3-trihydroxy-AQ), purpurin (1,2,4-trihydroxy-AQ), and anthrarufin (1,5-dihydroxy-AQ). Some hydroxy-AQ compounds exhibited activation by mammalian microsomal preparations, particularly at lower concentrations, and the majority of mutagenic hydroxy-AQ's appeared to revert strain TA 1537 (*his* 3076) specifically. The second involved frameshift mutagenesis by AQ compounds with primary amino and, in a few cases, with secondary amino groups. Mammalian microsomes invariable potentiated frameshift mutagenesis, and activity with strain TA 100 (sensitive to base pair substitution) is seen in a few cases, e.g., 1,2-diamino-AQ. The third involved AQ compounds with one or more nitro groups. These derivatives exhibited the least specificity with regard to the tester strain reverted and to microsomal activation. All seven nitro-AQs tested were mutagenic. In those compounds with ''mutagenic'' functional groups, the type of mutagenesis observed was usually $NO_2 > OH > NH_2$. AQs bearing halogens or sulfonate or alkyl groups were nonmutagenic, as were AQs substituted solely with secondary amines. (51 references)

4300 REMOVAL OF MINOR METHYLATION PRODUCTS 7-METHYLADENINE AND 3-METHYLGUANINE FROM DNA OF *ESCHERICHIA COLI* TREATED WITH DIMETHYL SULPHATE. (Eng.) Lawley, P. D. (Inst. Cancer Res., Royal Cancer Hosp., Pollards Wood Res. Station, Chalfont St. Giles, Bucks, HP8 4SP, U.K.); Warren, W. *Chem Biol Interact* 12(2): 211-220; 1976.

Persistence of methylpurines in DNA methylated in vitro and in vivo in *Escherichia coli* WP2 cells, by dimethyl sulphate (DMS) was studied, with particular reference to the minor products 7-methyladenine and 3-methylguanine known to be removed from DNA in vitro by spontaneous hydrolysis at neutral pH. The half-life of 7-methyladenine in vivo was relatively short (2.6 ± 0.2 hr) but not significantly shorter than in vitro at pH 7.2, 37 C. The half-life of 3-methylguanine was 3.6 ± 0.3 hr in vivo, markedly shorter than in vitro, where its stability was somewhat greater than that of 7-methylguanine. Enzymatic excision of 3-methylguanine was therefore indicated to occur in *E. coli*. Previous findings that 7-methylguanine is probably not enzymatically excised from DNA in vivo, whereas 3-methyladenine is rapidly removed, were confirmed, and additional support for the concept of enzymatic removal of 3-methyladenine was obtained by showing extensive inhibition of its removal from cells treated with iodoacetamide prior to methylation. It is suggested that methylations of adenine or guanine in DNA at N-3 constitute blocks to template activity of DNA and stimulate a ''repair'' response of enzymatic removal of 3-methylpurines. Ionized forms with positively charged amino groups may be the most effective blocks to template activity.

4301 FUKINOTOXIN, A NEW PYRROLIZIDINE ALKALOID FROM *PETASITES JAPONICUS*. (Eng.) Furuya, T. (Sch. Pharmaceutical Sciences, Kitasato Univ., Shirokane, Minato-ku, Tokyo 108, Japan); Hikichi, M.; Iitaka, Y. *Chem Pharm Bull* 24(5): 1120-1122; 1976.

In the course of studies on the carcinogenic activity of pyrrolizidine alkaloids in Compositae plants, a new alkaloid, fukinotoxin (I), which is highly cytotoxic, was isolated from the methanol extract of the young stalk of *Petasites japonicus* by silica gel column chromatography. Its stucture was shown to be (12R,13R,15R,3'R)-12-hydroxy-4,12,13-trimethyl-4,8-secosenec-1-enine-15-spiro-2'-(3'-methyl)oxiran with the aid of chemical and x-ray crystallographic analyses. The compound seems to be the main carcinogen in the young scape of these plants, recently considered a carcinogenic human food. The water solubility and structural conformation of fukinotoxin raise significant questions concerning its carcinogenesis. (8 references)

4302 INTERACTION OF AFLATOXIN B_1 WITH SOME BIOLOGICALLY IMPORTANT SUBSTANCES. (Eng.) Prasanna, H. R. (Vallabhbhai Patel Chest Inst., Univ. Delhi, Delhi, India); Gupta, S. R.; Viswanathan, L.; Venkitasubramanian, T. A. *Z Lebensm Unters Forsch* 160(4): 345-352; 1976.

The interaction of aflatoxin B_1 with a number of biologically important compounds was investigated by spectrophotometry and fluorimetry. The UV absorption spectra of tryptophan, adenosine, RNA, and DNA were altered by the addition of aflatoxin B_1, but RNA was the only one with increased absorption. This indicates that the mode of interaction between RNA and aflatoxin is different from that of the other compounds. Some amino acids, purine and pyrimidine derivatives, bovine serum albumin, and DNA quenched the fluorescence of aflatoxin B_1. Fluorescence polarization data suggest that the interaction of aflatoxin B_1 with bovine serum albumin was stronger than that with DNA.

4303 ACUTE AND PROTRACTED CHANGES IN THE LIVER OF SYRIAN HAMSTERS INDUCED BY A SINGLE DOSE OF AFLATOXIN B_1. OBSERVATIONS ON PATHOLOGICAL EFFECTS OF THE SOLVENT (DIMETHYLFORMAMIDE). (Eng.) Ungar, H. (Hadassah Medical Sch., Jerusalem, Israel); Sullman, S. F.; Zuckerman, A. J. *Br J Exp Pathol* 57(2): 157-164; 1976.

The pathological effects of a single ip injection of crystalline aflatoxin B_1 in solution with N-N-dimethylformamide (DMF) were investigated in male Syrian hamsters 8-10 wk old. The concentrations of aflatoxin were adjusted to the smallest controllable amount of solvent in relation to the size of the animal (0.5 or 1.0 ml/kg). Treatment with, respectively, 5.0 or 2.5 mg/kg aflatoxin in 1.0 ml/kg DMF was lethal to most animals within 1 wk. However, when the solvent was reduced to 0.5 ml/kg, although first week mortality was not eliminated entirely, survivors could be observed up to 28 days. Either dose of DMF alone was lethal for several animals during the first week and sufficient to produce histological lesions in the liver that were characteristic and clearly different from those induced by aflatoxin during the same period. DMF alone caused centrilobular necrosis that was accompanied by hemosiderosis of liver structures. With aflatoxin, in addition, periportal and midzonal necrosis of the liver was present. Aflatoxin B_1 and DMF both induce hemorrhagic tendency, probably by involvement in coagulation, but whether or not they act by a similar mechanism is not known. The results suggest that the Syrian hamster is more susceptible to the affects of aflatoxin B_1 than might be expected with estimated LD_{50}s of 10.2 mg/kg po and 6 mg/kg ip.

4304 IDENTIFICATION AND SEMIQUANTITATIVE DETERMINATION OF OCHRATOXINS A AND B, STERIGMATOCYSTIN AND AFLATOXIN B_1. (Pol.) Lemieszek-Chodorowska, K. (Zaklad Higieny, 00-791 Warsaw, ul. Chocimska 24, Poland). *Rocz Panstw Zakl Hig* 27(2): 153-160; 1976.

One-dimensional thin-layer chromatography was used for the identification and semiquantitative determination of aflatoxin B_1, sterigmatocystin, and ochratoxins A and B isolated in wheat, rye, and rice. The extracts were purified with the use of chromatographic columns filled with celite (for the ochratoxins) or silica gel (for aflatoxin B_1 and sterigmatocystin). The purified extracts were developed on the same chromatogram with a benzene + methanol + acetic acid (90 + 5 + 5) solvent system. Mycotoxin fluorescence was evaluated at a wavelength of 366 nm. Sterigmatocystin was visualized by spraying the chromatogram with an ethanol solution of $AlCl_3$, and an ethanol-water solution of $NaHCO_3$ was used as a spray to increase ochratoxin fluorescence. None of the mycotoxins were found in control tests. In approximately 74 trials fortified with the mycotoxins, mean values in the range 89.5%-92.9% were obtained. The Rf values obtained were: aflatoxin B_1 0.25-0.28; ochratoxin B 0.30-0.33; ochratoxin A 0.40-0.43; and sterigmatocystin 0.56-0.59. The lowest detectable amounts in the grain products were 0.001 μg for aflatoxin B_1, 0.0025 μg for ochratoxin A, 0.0025 μg for ochratoxin B, and 0.005 μg for sterigmatocystin. (20 references)

4305 EFFECT OF PHENOBARBITAL PRETREATMENT ON IN VITRO ENZYME KINETICS AND IN VIVO BIOTRANSFORMATION OF BENZENE IN THE RAT. (Eng.) Gut, I. (Center Industrial Hygiene and Occupational Diseases, 100 42 Praha 10, Srobarova 48, Czechoslovakia). *Arch Toxicol* 35(3): 195-206; 1976.

Phenobarbital pretreatment (50 mg/kg/day for 3 days orally) of male Wistar rats increased the Vmax of benzene hepatic microsomal biotransformation in vitro about sixfold without changing the Km. However, benzene blood levels after oral, ip, or sc benzene administration (3-3.5 mmoles/kg) were not influenced by phenobarbital pretreatment. The phenol blood levels after oral or ip benzene were increased by phenobarbital pretreatment, but less than expected from in vitro data and only 3 hr after benzene administration. Phenol elimination in urine after sc benzene was not affected by phenobarbital. After oral or ip benzene administration, phenol urine excretion closely followed the levels of phenol in blood; i.e., the rate of phenol urine excretion was significantly but shortly increased, and the cumulative urine excretion of phenol increased very little or remained unchanged. Differences between the in vitro and in vivo observations may be explained partly by: (a) distribution of benzene, which apparently limited benzene availability for biotransformation (Vd = 5.5) and caused rapid decrease of benzene concentrations in blood; (b) differing conditions for enzyme activity: in vitro concentrations of benzene were higher by at least an order of magnitude than phenol concentrations, while in vivo an opposite relation prevailed, making a competition for microsomal monooxygenase possible; and (c) cofactor availability, as benzene ring hydroxylation requires high energy. The rate of in vitro hepatic microsomal benzene biotransformation proved to be of limited value when predicting benzene quantitative biotransformation in vivo, in contradistinction to various substrates where the in vitro and in vivo biotransformation data are in good agreement. (19 references)

4306 SYNERGISTIC TOXICITY OF FOOD ADDITIVES IN RATS FED A DIET LOW IN DIETARY FIBER. (Eng.) Ershoff, B. H. (Inst. Nutritional Studies, Culver City, CA 90230). *J Food Sci* 41(4): 949-951; 1976.

Data are presented indicating that some food additives have a synergistic toxic effect when incorporated in a purified, low-fiber diet at dosages that have little if any adverse effect when fed individually. Immature male Sprague-Dawley rats (average initial body wt 50.4 g) were fed a purified, low-fiber diet to which were added supplements of 2% sodium cyclamate, 2% FD&C Red No. 2 (amaranth), and 4% polyoxyethylene (20) sorbitan monostearate (Tween 60) individually or in combination. Although supplements of any one of the three food additives had little if any deleterious effect, combined supplements of sodium cyclamate and FD&C Red No. 2 resulted in a marked retardation in weight gain, an unhealthy appearance of the fur, alopecia, and extensive diarrhea with watery and mushy stools. Concurrent administration of all three food additives resulted in further retardation in wt gain and death of all test animals within 14 days. The deleterious effects obtained on the latter diet could be largely counteracted by the concurrent administration of various dietary fiber-containing materials. Blond psyllium seed powder was particularly potent in this regard. The protective factor or factors in the powder were distinct from any of the known nutrients and from cellulose per se. The observation that some food additives have a cumulative or synergistic

ffect when incorporated in a purified, low-fiber
iet may be of considerable public health signifi-
ance. (12 references)

307 IN VITRO TRANSFORMATION OF NEWBORN HAMSTER
CELLS INDUCED BY SODIUM NITRITE. (Eng.)
suda, H. (Biological Res. Center, Central Res.
nst., Japan Tobacco and Salt Public Corporation, 23
akogi, Hatano, Kanagawa-ken 257, Japan); Inui, N.;
akayama, S. *Gann* 67(2): 165-173; 1976.

he in vitro carcinogenicity of sodium nitrite was
xamined. Addition of a high concentration of sodium
itrite (50 or 100 mM) to mass cultures of newborn
yrian hamster cells for 24 hr resulted in morpholog-
cal transformation of the cells in 21 days. In two
ontrol cultures, transformed cells appeared 10 wk
r more after those in treated cultures. Two of five
ransformed cultures produced progressively growing
ibrosarcoma tumors when they were injected into
oung adult hamsters. The chromosomes were analyzed
t several stages of transformation: in the early
tages, the chromosome number varied widely from
ear diploid to tetraploid, but later they became
ear diploid. Morphological alteration caused by
odium nitrite was also seen in a short-term assay
n which hamster embryonic cells (1 x 10^4 cells/60-
m dish) were treated. The alteration occurred 8
ays after treatment. (30 references)

308 TUMOR-PROMOTING ACTIVITY OF CERTAIN EXTRACTS
OF TOBACCO. (Eng.) Chortyk, O. T. (Tobacco
ab., Agricultural Res. Service, U.S. Dept. Agricul-
ure, Athens, GA 30604); Bock, F. G. *J Natl Cancer
nst* 56(5): 1041-1045; 1976.

he tumor-promoting activities of extracts of flue-
ured and cigarette tobacco were evaluated. Initially,
lue-cured tobacco was extracted consecutively with
exane, chloroform, acetone, ethyl alcohol, methyl
lcohol, and water. The extracts were tested for
umor-promoting activity on the skin of female ICR
wiss mice. The hexane and chloroform extracts were
ractionated on silicic acid. These fractions were
evoid of tumorigenic activity, and the acetone and
lcohol extracts showed only marginal activity.
ubsequently, chloroform-extracted cigarette tobacco
as extracted with water, and the aqueous extract
as partitioned by solvent precipitation methods.
ioassay results showed tumor-promoting activity for
he aqueous extract, with tumors in 38% of the ani-
als. The aqueous extract appeared about five times
s active as the smoke condensate derived from an
qual weight of tobacco. (23 references)

309 N'-NITROSONORNICOTINE IN JAPANESE TOBACCO
PRODUCTS. (Eng.) Bharadwaj, V. P. (Dept.
xperimental Pathology, Cancer Inst., Kami-Ikebukuro
-37-1, Toshima-ku, Tokyo 170, Japan); Takayama, S.;
amada, T.; Tanimura, A. *Gann* 66(5): 585-586; 1975.

apanese commercial tobacco products were analyzed
or N'-nitrosonornicotine (NNN), a compound that
eportedly has induced multiple pulmonary adenomas
the lung and bronchi of mice after ip administration and esophageal tumors in rats when given in drinking water. Starting with 50 g of tobacco, chloroform extracts were dried, and a residue of the basic fraction was chromatographed over a Florisil column and eluted with 100 ml of CH_2Cl_2 followed by elution with 100 ml of CH_2Cl_2-acetone (9:1). CH_2Cl_2-acetone eluates were collected and evaporated, and the residue was dissolved in 1 ml of acetone. Gas chromatographic analyses of six brands of cigarettes, three types of pipe tobacco, and a Kiseru tobacco (fresh leaves dried without fermentation) revealed an NNN level of 1.1 ppm in one of the cigarette products and an NNN level of 1.8 ppm in Kiseru tobacco. The presence of NNN was either doubtful or not detected in the other products analyzed. (10 references)

4310 CANCER AND CONGENITAL ANOMALIES ASSOCIATED WITH ANESTHETICS. (Eng.) Corbett, T. H. (US Veterans Admin. Hosp., Ann Arbor, MI 48105). *Ann NY Acad Sci* 271: 58-66; 1976.

Certain anesthetics in general use may be carcinogenic, embryolethal, teratogenic, and/or mutagenic. Spontaneous abortion rates as high as 38% have been reported among nurse anesthetists, compared to a rate of 9% among general duty nurses, and 16.4% of 434 children born to nurse anesthetists who had worked during pregnancy had birth defects, compared to only 5.7% of 261 children born to nurse anesthetists who had not worked while pregnant. Three studies have indicated an increased cancer risk in operating-room personnel; a recent survey involving 50,000 such personnel and 30,000 non-operating room female medical personnel controls revealed an increased prevalence of cancer in the former, ranging from 1.3 to 2 times the control rate. Halogen ether anesthetics such as isoflurane, methoxyflurane, and enflurane are similar in chemical structure to the proved carcinogen bis(chloromethyl) ether, and trichloroethylene is likewise similar to the carcinogen vinyl chloride. Isoflurane has been shown to be carcinogenic in mice. When pregnant Swiss/ICR mice were exposed to 0.5% isoflurane for 2 hr on days 12, 14, 16, and 18 of pregnancy and the offspring were exposed to 0.1% isoflurane every other day from age 5 days for 25 exposures, 10/37 male offspring killed after 15 mo had hepatic neoplasms, with three animals having multiple tumors. No neoplasms were observed among 23 control animals. Isoflurane, and the structurally similar halogenated ether and alkane anesthetic agents, must be regarded with suspicion and studied for carcinogenic properties as soon as possible. (32 references)

4311 MUTAGENICITY AND PUTATIVE CARCINOGENICITY TESTS OF SEVERAL POLYCYCLIC AROMATIC COMPOUNDS ASSOCIATED WITH IMPURITIES OF THE INSECTICIDE METHOXYCHLOR. (Eng.) Grant, E. L. (Dept. Biology, Univ. Victoria, Victoria, B. C., V8W 2Y2, Canada); Mitchell, R. H.; West, P. R.; Mazuch, L.; Ashwood-Smith, M. J. *Mutat Res* 40(3): 225-228; 1976.

Several polycyclic hydrocarbons, 3,6-dimethoxy-9,10-bis(p-methoxyphenyl)phenanthrene, tetrakis(p-methoxyphenyl)ethylene, and 3,6,11,14-tetramethoxydibenzo(g,p)chrysene, associated as impurities in commercial samples of the insecticide methoxychlor, were tested by the Ames mutagenicity test with strains of *Salmonella typhimurium*, TA 1535, TA 1537, TA 1538, and TA 98. Activation by liver microsomes induced with either phenobarbitol or a polychlorinated biphenyl mixture (Aroclor) was examined. The only active compound was 3,6,11,14-tetramethoxydibenzo(g,p)chrysene, mutagenic (0.39 revertant/nmol) to strain TA 98. The results are of interest in terms of the widespread use of methoxychlor. (7 references)

4312 ELIMINATION OF AZIRIDINE RESIDUES FROM CHEMOSTERILISED MOSQUITOES. (Eng.) Sharma, V. P. (Vector Control Res. Center, Indian Council of Medical Res., 22 Sham Nath Marg, Delhi--110054, India). *Nature* 261(5556): 135; 1976.

Aziridine residues were eliminated from mosquitoes chemosterilized with triethylene thiophosphoramide (thiotepa) by dipping the sterile insects into acid and then alkaline solutions. One hundred mosquito pupae were placed in a 0.6% thiotepa solution containing 0.01 N H_2SO_4 (pH 1.8-2.0) for 3 hr, and pH 3 was maintained by addition of small increments of H_2SO_4 for 90 min. At emergence, an equal number of virgin females was introduced into the cages, and egg rafts were collected for routine bioassay. Breakdown of thiotepa to ethyleneimine was complete in acid solution. As previously reported, dipping acidified pupae in alkaline solution (pH 9) accelerated the conversion of ethyleneimine to deuteriophosphoric acid and D_2NCH_2-CH_2OD, making the mosquitoes free of any traces of aziridine compounds. These treatments had no effect on the longevity or mating competitiveness of the male mosquitoes. The results indicate that mass release of sterilized male mosquitoes treated in this manner would not be hazardous to the food chain and would not contaminate the environment directly or indirectly.

4313 COMPARATIVE ADDUCT FORMATION OF 4-AMINOBIPHENYL AND 2-AMINOFLUORENE DERIVATIVES WITH MACROMOLECULES OF ISOLATED LIVER PARENCHYMAL CELLS. (Eng.) King, C. M. (Michael Reese Hosp. Medical Center, Chicago, Il 60616); Traub, N. R.; Cardona, R. A.; Howard, R. B. *Cancer Res* 36(7): 2374-2381; 1976.

Isolated rat liver parenchymal cells from adult Sprague-Dawley rats have been used in a study of the metabolic activation of derivatives of the carcinogens 4-aminobiphenyl and 2-aminofluorene. The formation of adducts of these compounds with cellular RNA and protein has been taken as evidence of their transformation to metabolites that are capable of spontaneous reaction with tissue macromolecules. The hydroxamic acid N-hydroxy-N-4-acetylaminobiphenyl (N-hydroxy-AABP) was bound to RNA to a greater extent than were the amino-, hydroxylamino-, nitroso-, nitro-, acetylamino-, or azoxybiphenyl derivatives. RNA adducts of the hydroxamic acid retained little of the acetyl group. The structural requirements for binding and the nature of the bound derivatives are consistent with the activation of N-hydroxy-AABP by N → O acyltransfer. Approximately equal quantities of 4-nitrosobiphenyl and the hydroxamic acid were bound to protein, but far less of the nitroso derivative was incorporated into RNA. Adduct formation of N-hydroxy-N-2-acetylaminofluorene with RNA occurred with retention of the acetyl group and was dependent on the sulfate concentration in the media. Consequently, reaction of the fluorenyl derivative with RNA probably resulted from conjugation of the hydroxamic acid with sulfate. (47 references)

4314 MUTAGENESIS AND CARCINOGENESIS. (Eng.) Boyland, E. (London Sch. Hygiene and Tropical Medicine, Keppel St. Gower St., London WC1E 7T, England). *Arch Geschwulstforsch* 45(7): 625-627; 1976.

The relation between mutagenesis and carcinogenesis is discussed. Comparisons of induction of cancer and mutation are difficult because carcinogenesis is generally studied in vertebrates while mutagenesis is generally studied in invertebrates. Because it is necessary to change the germ cells to induce heritable mutations, strict comparison of mutagenesis and carcinogenesis requires that only compounds that induce cancer of the germ tissues be used. Following the production of the primary biochemical lesion in the nucleic acid, the cells will either be repaired, or they will divide with the altered nuclear material and produce mutations; the chance of mutation or carcinogenesis therefore depends upon the balance of the damaging processes and the repair processes.

4315 ALTERATIONS IN THE TRANSCRIPTIONAL CAPACITY OF HEPATIC DNA-DEPENDENT RNA POLYMERASE I AND II BY N-HYDROXY-2-ACETYLAMINOFLUORENE. (Eng.) Glazer, R. I. (Dept. Pharmacology, Emory Univ., Atlanta, GA 30322). *Cancer Res* 36(7): 2282-2289; 1976.

This study was designed to measure the ability of RNA polymerases I and II to copy faithfully both the natural and synthetic DNA templates as well as those that had been reacted with N-2-acetoxy-2-acetylaminofluorine. With the use of hepatic RNA polymerases I and II, acetylaminofluorene modification of DNA in vitro results in reduction of RNA elongation but not in an altered frequency of incorporation of (α-^{32}P)-ribonucleotides. In contrast, in vivo modification of RNA polymerases by a single dose of N-2-hydroxy-2-acetylaminofluorene (N-OH-AAF) results in an alteration of their capacity for transcribing a normal RNA product but does not affect the product size transcribed from normal or carcinogen-modified templates. The dose and sex dependency of the ability of N-OH-AAF to modify in vivo RNA polymerases is noted; e.g., higher doses are required to inhibit RNA polymerase activities and nRNA synthesis in female rats. N-OH-AAF is also target specific, RNA polymerase II being considerably more sensitive than RNA polymerase I. It is not known by what method enzyme modification by N-OH-AAF occurs. Nearest-neighbor analyses show an altered distribution of (α-^{32}P)-cytidine monophosphate and (α-^{32}P)-guanosine monophosphate in the RNA transcribed by the N-OH-AAF-modified polymerases, implying that an unequal copying of complementary sequences in the DNA occurs by the modified enzyme. This perturbation in information flow may represent a mechanism for inducing mutagenic changes in RNA without modifications in DNA. (28 references)

4316 STUDIES ON THE MECHANISM OF INHIBITION OF 2-ACETYLAMINOFLUORENE TOXICITY BY BUTYLATED HYDROXYTOLUENE.

(Eng.) Goodman, J. I. (Dept. Pharmacology, Michigan State Univ., East Lansing, MI 48824); Trosko, J. E.; Yager, J. D., Jr. *Chem Biol Interact* 12(2): 171-182; 1976.

Male rats were placed on a diet containing 0.05% (w/w) of the hepatic carcinogen 2-acetylaminofluorene (AAF). They ceased to gain weight. However, when the carcinogenic diet was supplemented with butylated hydroxytoluene (BHT) (0.5% w/w), an antioxidant, the animals gained weight at approximately one-half the normal rate. Studies were directed towards the effect of BHT on the extent and duration of the covalent binding of AAF with DNA. BHT feeding was shown to reduce the binding of carcinogen to hepatic DNA. Studies with cultured cells demonstrated that BHT does not influence either excision repair or post-replication repair of DNA. These data indicate that a potential mechanism of action of BHT is at the anti-initiation level of carcinogen-induced DNA damage.

4317 INCREASED SUSCEPTIBILITY TO CARCINOMA OF THE LIVER IN RATS WITH ONE KIDNEY INGESTING N-4-(4'-FLUOROBIPHENYL)ACETAMIDE.

(Eng.) Reuber, M. D. (11014 Swansfield Road, Columbia, MD 21044). *Gann* 67(2): 307-313, 1976.

The role of the kidneys in hepatic carcinogenesis was studied in inbred Buffalo strain male rats ingesting 0.04% N-4-(4'-fluorobiphenyl)acetamide (4'-F-4BAA) in the diet. Experimental groups were made up of male rats with both kidneys intact and male rats with the left kidney removed. Carcinomas of the liver developed in all 19 rats with uninephrectomy, and the number of rats with large carcinomas, multiple carcinomas, poorly differentiated and undifferentiated carcinomas, and metastases was also greater. Apparently the animals with one kidney removed were unable to secrete the metabolites of 4'-F-4BAA as readily as the rats with both kidneys. Metabolites are then able to erect an additional effect on the liver and increase the development of carcinomas of the liver. (17 references)

4318 INFLUENCE OF BLOOD COLLECTION ON INCIDENCE OF 2,7-FAA INDUCED RAT LEUKEMIA.

(Eng.) Fujiwara, M. (Tokyo Medical and Dental Univ., Tokyo, Japan); Takayama, S. *Acta Pathol Jpn* 26(2): 139-145; 1976.

The influence of blood collection on induced leukemia in 3-wk-old Wistar rats was studied in seven experiments in which conditions varied. N,N'-2,7-Fluorenylenebisacetamide was administered po in a solid diet that contained 0.025% of the carcinogen; leukemia, especially granulocytic leukemia, was induced. The peripheral blood was examined on various schedules, one to four times a wk for 9-16 wk. The blood volume lost by a single collection was small, but it was not neglible in small animals such as rats when the loss was repeated. The average total volume of blood loss in the seven experiments ranged from 0.12 to 0.63 ml. There was a positive correlation between the incidence of leukemia and the volume of blood lost ($r = 0.92$; $0.01 > p > 0.001$). The induction of mature granulocytic leukemia was thought to be increased by the promotion of the granulopoiesis that had been suppressed by 2,7-FAA. It was concluded that the blood loss by repeated blood collection for examination raised the incidence of 2,7-FAA induced leukemia.

4319 ACCELERATION OF HEPATOCARCINOGENESIS OF 2,7-BIS(ACETAMIDO)FLUORENE BY CARBON TETRACHLORIDE AND TIME RELATION OF TREATMENT.

(Eng.) Kozuka, S. (Nagoya Univ. Sch. Medicine, 65 Tsurumai-cho, Showa-ku, Nagoya 466, Japan); Sassa, R. *Gann* 67(2): 141-145; 1976.

The role of the time between administration of CCl_4 and feeding of 2,7-bis-(acetamido)fluorene (2,7-AAF) in the hepatocarcinogenesis of SMA/M_5 mice was analyzed in 14 groups with different feeding schedules. Simultaneous treatment with CCl_4 and either continuous or intermittent feeding of 2,7-AAF increased the average number of hepatic nodules and the incidence of hepatomas, while 8 wk of feeding of 2,7-AAF followed by 8 wk of treatment with CCl_4 (and vice versa) did not accelerate hepatocarcinogenesis. Alternating treatments increased the average number of hepatic nodules but failed to induce hepatomas. Allyl alcohol also failed to accelerate 2,7-AAF hepatocarcinogenesis. Intermittent 1-wk feeding of 2,7-AAF with 3-wk intervals decreased the average number of hepatic nodules, compared with continuous feeding of the carcinogen. (25 references)

4320 EFFECT OF AVITAMINOSIS A AND HYPERVITAMINOSIS A ON URINARY BLADDER CARCINOGENICITY OF N-[4-(5-NITRO-2-FURYL)-2-THIAZOLYL]FORMAMIDE.

(Eng.) Cohen, S. M. (Univ. Wisconsin Center Health Sciences, Madison, WI 53706); Wittenberg, J. F.; Bryan, G. T. *Cancer Res* 36(7): 2334-2339; 1976.

The effect of vitamin A deficiency and hypervitaminosis A on the urothelial carcinogenicity of N-[4-(5-nitro-2-furyl)-2-thiazolyl]formamide (FANFT) was determined in female weanling Sprague-Dawley rats. Vitamin A deficiency resulted in squamous metaplasia of the urinary bladder and high incidences of cystitis, ureteritis, and pyelonephritis. Administration of FANFT to vitamin A-deficient rats appeared to accelerate the carcinogenicity, with earlier appearance of urinary bladder tumors and the development of ureteral and renal pelvic carcinomas. Most of these tumors were squamous cell foci. Hypervitaminosis A prevented the appearance of squamous metaplasia and squamous cell neoplasia in rats fed FANFT, but it did not inhibit the formation of transitional cell hyperplasia or neoplasia in comparison to rats receiving normal levels of vitamin A and FANFT. The dosage and form of vitamin A, found to have contradictory effects in a number of studies, are suggested as an explanation for these findings. (30 references)

4321 SQUAMOUS METAPLASIA OF THE TRACHEAL EPITHELIUM IN ORGAN CULTURE. I. EFFECTS OF HYDROCORTISONE AND β-RETINYL ACETATE. (Eng.) Mossman, B. T. (Coll. Medicine, Univ. Vermont, Burlington, VT 05401); Ley, B. W.; Craighead, J. E. *Exp Mol Pathol* 24(3): 405-414; 1976.

Organ cultures of hamster trachea from inbred female golden Syrian hamsters were incubated up to 16 wk in chemically defined medium containing 50 μg/ml of hydrocortisone sodium succinate or the vitamin A analog, β-retinyl acetate. Medium from replicate cultures was assayed weekly for acid phosphatase activity, and representative tissues were harvested after pulse labeling with ^{3}H-thymidine. Explants maintained in the medium containing hydrocortisone showed basal cell proliferation, squamous metaplasia, and extensive keratinization, whereas those in the medium containing β-retinyl acetate exhibited a proliferative ciliated epithelium that gradually became hypoplastic after 8 wk in vitro. Simultaneous addition of these agents in equal concentrations resulted in an epithelium that appeared dedifferentiated. Acid phosphatase activity in cultures containing hydrocortisone was elevated. In contrast, release of the enzyme was inhibited when β-retinyl acetate was added to the culture medium. Results indicate that hydrocortisone and β-retinyl acetate act in a competitive fashion in basal cell differentiation. (35 references)

4322 LIVER-CELL ADENOMAS AND ORAL CONTRACEPTIVES (Letter to Editor). (Eng.) Sorensen, T. I. A. (Gentofte Hosp., Copenhagen, Denmark). *N Engl J Med* 294(19): 1061; 1976.

The possibility of case selection bias in a study associating oral contraceptives with liver cell adenoma is discussed. Cases received from other institutions after 1973, the time at which this association was first presented in the literature, were included, and may have selectively increased the number of reported tumors in women presenting long-term use of oral contraceptives.

4323 LIVER-CELL ADENOMA AND ORAL CONTRACEPTIVES (Letter to Editor). (Eng.) Edmondson, H. A. (Univ. Southern California, Los Angeles, CA); Henderson, B. E.; Benton, B. *N Engl J Med* 294(19): 1064; 1976.

In response to several letters criticizing their selection of controls, the authors of a report associating mestranol (as opposed to ethinyl estradiol) with liver cell adenoma reanalyzed their data. However, this re-evaluation led to the same conclusion expressed in their paper: that the type of synthetic estrogen used in the oral contraceptive seems to be an important determinant of risk. Patients who developed liver cell adenoma had used mestranol-containing compounds almost exclusively.

4324 MENOPAUSAL ESTROGENS AND BREAST CANCER. (Eng.) Hoover, R. (Environmental Epidemiology Branch, NCI, Room C525, Landow Bldg., Bethesda, MD 20014); Gray, L. A.; Cole, P.; MacMahon, B. *N Engl J Med* 295(8): 401-405; 1976.

A total of 1,891 women (from one private practice in Louisville, KY) who had been treated with conjugated estrogens, po for at least 6 mo were followed for 12 yr (mean) for incidence of breast cancer. Overall, 49 cases were observed; 39.1 were expected on the basis of rates in the general population (relative risk = 1.3). The relative risk increased with follow-up duration, progressing to 2.0 after 15 yr (13/6.6). The excess risk after 10 yr was not due simply to prolonged estrogen use, since there was no clear dose-response relation to accumulated years of use. However, higher risk accrued to women using higher-dose tablets and those taking the medication on an other than daily basis. In addition, after 10 yr of follow-up, two factors related to low risk of breast cancer, multiparity and oophorectomy, were no longer so related. Finally, estrogen use was related to an especially high risk of breast cancer among women in whom benign disease developed after they had started the drug. (33 references)

4325 EFFECT OF TAMOXIFEN (ICI 46,474) ON INITIATION AND GROWTH OF DMBA-INDUCED RAT MAMMARY CARCINOMATA. (Eng.) Jordan, V. C. (Sch. Medicine, Leeds LS2 9NL, England). *Eur J Cancer* 12(6): 419-424; 1976.

Single large doses of tamoxifen, which produce prolonged antiestrogen effects, were administered to 50-day-old female Sprague-Dawley rats to inhibit the initiation and growth of rat mammary tumors induced by 7,12-dimethylbenz(a)anthracene (DMBA). Tamoxifen (5 mg/day x 2) administered sc at the time of DMBA administration reduced the number of mammary tumors found 120 days later to less than 10% of controls. A second dose regimen of tamoxifen 30 days after DMBA inhibited tumorigenesis up to 120 days. Tamoxifen (5 mg/day x 2) administered to rats with established mammary tumors produced antitumor effects for 3 wk. Treatment tumors were significantly smaller than controls. The binding of ^{3}H-estradiol-17β in vivo was significantly lower in the uterine, vaginal, and mammary tumors of treated animals compared with controls. Tamoxifen also inhibited estrogen-stimulated elevations in plasma prolactin and uterine wet weights in ovariectomized rats. The inhibition of estrogen binding in the tumors is probably of fundamental importance in the antitumor effects of tamoxifen since it produced only partial inhibition of prolactin release. (27 references)

4326 PROLACTIN AND ESTROGEN DEPENDENCY OF RAT MAMMARY CANCERS AT EARLY AND LATE STAGES OF DEVELOPMENT. (Eng.) Bradley, C. J. (Dept. Physiology, Michigan State Univ., East Lansing, MI 48824); Kledzik, G. S.; Meites, J. *Cancer Res* 36(2): 319-324; 1976.

Estrogen and prolactin dependency were separated in 7,12-dimethylbenz(a)anthracene (DMBA)-induced rat mammary tumors at 2.5 and 5 mo after DMBA injection. Ovariectomy and drug and/or hormone treatments were used to produce an estrogen or prolactin deficiency for two weeks, followed by a two-week period in which the deficiency was corrected. Tumors were classified as estrogen or prolactin-dependent based upon regression in the absence of the hormone and resumption of growth upon hormone replacement. At 2.5 mo and 5 mo after DMBA injection, about 29 and 33% of the tumors, respectively, were classified as prolactin-dependent, and 35 and 45%, respectively, were classified as estrogen-dependent. However, the percentage of estrogen-dependent tumors was reduced to 2.2 and 9.7%, respectively, when prolactin levels were maintained after ovariectomy. These results indicate that most DMBA-induced mammary tumors in Sprague-Dawley female rats are dependent on both estrogen and prolactin but that ovariectomy or estrogen administration do not accurately reflect estrogen dependency, since prolactin secretion also is altered by these procedures.

4327 COMPARISON OF THE PRODUCTS OF THE REACTION OF 7-METHYLBENZ[a]ANTHRACENE 5,6-OXIDE AND RNA, WITH THOSE FORMED IN 7-METHYLBENZ[a]ANTHRACENE-TREATED CELLS. (Eng.) Baird, W. M. (Wistar Inst., 36th St. at Spruce, Philadelphia, PA 19104); Grover, P. L.; Sims, P.; Brookes, P. *Cancer Res* 36(7): 2306-2311; 1976.

RNA was isolated by a phenol extraction method from C57BL mouse embryo cells treated in culture with either (G-^{3}H)-7-methylbenz(a)anthracene (7-MeBA) or (G-^{3}H)-7-methylbenz(a)anthracene 5,6-oxide (7-MeBA-epoxide, the K-region epoxide). The RNA was degraded to ribonucleosides, mixed with UV-absorbing quantities of the epoxide ribonucleoside products isolated from RNA that had reacted with 7-MeBA-epoxide in aqueous ethanol solution, and chromatographed on a column of Sephadex LH-20 eluted with a methanol: water gradient. The 7-MeBA-epoxide ribonucleoside products formed in cells were identical to those formed in aqueous solution, although the relative amounts varied. The majority of these epoxide-ribonucleoside products were not identical to those formed in cells treated with the parent hydrocarbon. These results suggest that the major reactive form of 7-MeBA that binds to RNA in mouse embryo cells is not the K-region epoxide of this hydrocarbon. Recent studies with hamster embryo cells suggest that benz(a)anthracene and benzo(a)pyrene become bound to DNA through a non-K-region dihydrodiol-epoxide; a similar process could be involved in the metabolic activation of 7-MeBA. (21 references)

4328 MECHANISM OF THE PENETRATION OF CARCINOGENIC POLYCYCLIC HYDROCARBONS THROUGH THE PLACENTA IN RATS AND MICE. (Rus.) Baranova, L. N. (N. N. Petrov Scientific Res. Inst. Oncology, Leningrad, USSR); Shendrikova, I. A.; Aleksandrov, V. A.; Likhachev, A. Ia.; Ivanov-Galitsin, M. N.; Dikun, P. P.; Napalkov, N. P. *Dokl Akad Nauk SSSR* 228(3): 733-736; 1976.

The time of concentration of polycyclic aromatic hydrocarbons in rat and mice fetuses was studied mathematically under specific initial assumptions concerning the transplacental passage of these compounds. Theoretical and experimental data were compared. Determination of the time of concentration in the maternal liver and in the fetuses of pregnant rats and mice inoculated in a single dose iv with 9,10-dimethyl-1,2-benzanthracene (DMBA) and 3,4-benzpyrene (BP) showed that placental diffusion takes place significantly more slowly than diffusion in the mother or fetus. Also, the polycyclic aromatic hydrocarbon concentration in the mother decreases exponentially, but the increase in concentration in the fetuses is delayed compared with maternal level. For a defined time interval, the concentration of the carcinogen decreased in the maternal rat and rose in the fetuses. Carcinogenic concentrations disappeared more rapidly from maternal mice than from their rat counterparts. The finding of a slower decrease in the rat fetuses than in the mother was not inconsistent with the mechanism of simple diffusion as the basis of transplacental passage of polycyclic aromatic hydrocarbons in rats. The metabolic rate in the mother determined the concentration of polycyclic aromatic hydrocarbons in the fetuses and the regularities of the changes. (14 references)

4329 CELLULAR KINETICS AND HISTOLOGICAL CHANGES IN EXPERIMENTAL CANCER OF THE UTERINE CERVIX. (Eng.) Hasegawa, K. (Dept. Obstetrics and Gynecology, Kobe Univ. Sch. Medicine, Ikuta-Ku, Kobe 650, Japan); Matsuura, Y.; Tojo, S. *Cancer Res* 36(2): 359-364; 1976.

Carcinoma of the mouse uterine cervix was induced by the insertion of 20-methylcholanthrene (MC) thread into the uterine cavity. Through biweekly observations, it was seen that the histopathological carcinogenesis in the cervix was characterized by three distinct changes: normal epithelium, anaplastic epithelium, and carcinoma. The incidence rate of normal epithelium declined sharply in the first four weeks, followed by slight decline up to the 20th wk of observation. Nevertheless, about 20% of MC-treated mice maintained histologically normal epithelium even at the 20th wk. In contrast, the incidence rate of anaplastic epithelium increased sharply in the first four weeks and thereafter tended to decline. Carcinoma was first observed at the fourth week, and its incidence rate increased thereafter almost linearly, reaching about 50% of the MC-treated mice at the 20th wk. Carcinoma was divided into two groups, the early invasive and the frank invasive types. The former was further subgrouped into epithelial bud, nodular growth, epithelial cord, and mixed type by the mode of stromal invasion. Studies using [^{3}H]-thymidine autoradiography on the cell proliferation kinetics in the process of carcinoma development showed that a labeling index became higher as the malignant changes of the epithelium advanced. Moreover, S-phase prolongation was observed with the malignant changes, and the cell cycle time did not differ markedly among normal epithelium (22.8 hr), anaplastic epithelium (23.0 hr), and frank invasive carcinoma (26.1 hr). However, the growth fraction was greatly varied; it was 28% for normal epithelium, 61% for anaplastic epithelium, 100% for early invasive carcinoma, and 100% for frank invasive carcinoma. These results indicate that the growth fraction increases with the malignant changes in the MC-treated mouse uterine cervix.

4330 THE BIOCHEMISTRY OF PRENEOPLASIA IN MOUSE SKIN. (Eng.) Boutwell, R. K. (McArdle Lab. Cancer Res., Univ. Wisconsin, Madison, WI 53706). *Cancer Res* 36(7): 2631-2635; 1976.

The early biochemical changes that occur in a two-stage (initiation and promotion) model system for skin (mouse skin) are described and background experiments are discussed. It was shown that a complete carcinogen for the skin, 7,12-dimethylbenz(a)anthracene (DMBA), provides both initiating and promoting components, but as the dose level is lowered, its promoting activity is lost. At the dose level that is completely carcinogenic (25 μg/wk), DMBA exhibits the properties of a promoter; it stimulates RNA, protein, and DNA synthesis followed by cell division. At initiating doses (2.5 and 0.25 μg/wk), both the biochemical and morphological (increased mitosis) responses to treatment that are characteristic of promoters are lost; the remaining biochemical effects involve DNA and are attributed to initiating action. Data supporting the concept that gene activation is involved in tumor promotion are also reviewed. (27 references)

4331 THE EFFECTS OF DEXAMETHASONE ON MOUSE SKIN INITIATION AND ARYL HYDROCARBON HYDROXYLASE. (Eng.) Thompson, S. (Fred Hutchinson Cancer Res. Center, 1102 Columbia St., Seattle, WA 98104); Slaga, T. J. *Eur J Cancer* 12(5): 363-370; 1976.

Mouse epidermis contains a NADPH-dependent aryl hydrocarbon hydroxylase (AHH) that is inducible by polycyclic hydrocarbons. The effects of dexamethasone, an anti-inflammatory steroid, on skin carcinogenesis and on mouse epidermal AHH from female Charles River CD-1 mice were investigated. Dexamethasone reduced tumor initiation by both 3-methylcholanthrene (MC) and 7,12-dimethylbenz(a)anthracene. With MC, dexamethasone caused at least a 50% reduction in both percentage of animals with tumors and number of papillomas per mouse. The mechanism by which dexamethasone and other glucocorticoids inhibit tumor production may be related to their ability to inhibit DNA synthesis, which consequently counteracts the induction of hyperplasia by tumor promoters. Dexamethasone applied topically to mice at a dose level of 25 or 75 μg slightly stimulated the AHH activity in mouse epidermis with peaks of 150% or 180% of controls, respectively, at 2 hr and a return to control levels by 4 hr. This was followed by a weak inhibition between 4 and 8 hr after treatment with either dose of dexamethasone. Dexamethasone did not inhibit benz(a)anthracene-induced AHH activity in vitro when added to the assay, even at concentrations greater than equimolar amounts of benzo(a)pyrene. However, if dexamethasone or fluocinolone acetonide were applied topically with MC, they inhibited the MC induction of AHH by at least 50% between 4 and 18 hr after simultaneous treatment. The more potent anti-inflammatory steroids, fluocinolone acetonide and fluclorolone acetonide, had a greater inhibitory effect on the MC induction of AHH after simultaneous treatment. The mouse skin tumor promoters, croton oil and 12-O-tetradecanoylphorbol-13-acetate, had only a weak stimulatory effect on AHH activity. The mechanism by which the anti-inflammatory steroids inhibit tumor initiation may be related to their ability to inhibit the metabolism of polycyclic hydrocarbons to their ultimate carcinogenic form(s). (25 references)

4332 RE: EARLY NEOPLASIA OF RABBIT PANCREATIC DUCTAL CELLS INDUCED BY DIMETHYLHYDRAZINE (Letter to Editor). (Eng.) Pour, P. (Univ. Nebraska Medical Center, Omaha, NB 68105). *Cancer Res* 36(5): 1848; 1976.

It is suggested that the ''early, well-differentiated carcinomas'' illustrated in a previous paper on the effect of dimethylhydrazine on rabbit pancreatic tissue is nothing more than mild hyperplasia. Neither atypical cells nor invasion was observed.

4333 NULL CARCINOGENIC EFFECT OF LARGE DOSES OF NITROSOPROLINE AND NITROSOHYDROXYPROLINE IN WISTAR RATS. (Eng.) Nixon, J. E. (Dept. Food Science and Technology, Oregon State Univ., Cornvallis, OR 97331); Wales, J. H.; Scanlan, R. A.; Bills, D. D.; Sinnhuber, R. O. *Food Cosmet Toxicol* 14(2): 133-135; 1976.

The carcinogenic activity of nitrosoproline and nitrosohydroxyproline in rats given large doses over a short period of time was studied. The nitrosamino acids were given po to weaning male, Wistar rats in four weekly doses totaling 290 mg/animal and the rats were observed over their lifetime. Negative controls received water and positive controls were given diethylnitrosamine. The rats were maintained on a basal semipurified diet or a basal diet containing cyclopropenoid fatty acids. No tumorigenic effect was seen in rats given nitrosoproline or nitrosohydroxyproline. Diethylnitrosamine produced a high incidence of hepatomas, as expected. Cyclopropenoid fatty acids had no significant effect on the carcinogenicity of the nitrosamino acids. This result is not surprising since cyclopropenoid fatty acids may exert a cocarcinogenic effect and the nitrosamino acids did not show any carcinogenic activity.

4334 STUDIES ON NITROSAMINE METABOLISM. I. SUBCELLULAR DISTRIBUTION OF RADIOACTIVITY IN TUMOR-SUSCEPTIBLE TISSUES OF RFM MICE FOLLOWING ADMINISTRATION OF [^{14}C]DIMETHYLNITROSAMINE. (Eng.) Daugherty, J. P. (Univ. Tennessee-Oak Ridge Graduate Sch. Biomedical Sciences, Oak Ridge, TN 37830); Clapp, N. K. *Life Sci* 19(2): 265-271; 1976.

The distribution of radioactivity in tumor-susceptible (liver and lung) and non-tumor-susceptible (heart, forestomach, and esophagus) tissues of male RFM mice was investigated at timed intervals following a single intragastric administration of ^{14}C-dimethylnitrosamine (DMN). Maximum radioactivity was associated with the tumor-susceptible tissues--liver and lung. At 15 min, the relative amount of radioactivity in the homogenates of heart, forestomach, esophagus, liver, and lung was 1, 2, 3, 10, and 70, respectively. The acid-soluble components of lung contained about six times as much radioactivity as the liver 15 min after administration; at 16 hr, the level of radioactivity had decreased and was equal in amount. The acid-insoluble components of both tumor-susceptible tissues incorporated much less radioactivity than the acid-soluble components, indicating that only a small amount of methyl label is covalently bound to cellular macromolecules. The amount of radioactivity in the acid-insoluble components ranged from 2%-34% in the lung and from 11%-33% in the liver. In the lung cytosol fraction the range of radioactivity was 75%-89% for the acid-soluble components of liver cytosol fraction ranged from 50%-89%, and from 52%-68% for the acid-insoluble components. The results suggest differences in the affinity, transport, and/or metabolism of DMN between liver and lung, as well as between tumor-susceptible and non-tumor-susceptible tissues. (27 references)

4335 THE ZONAL DISTRIBUTION OF THE HAPTOGLOBIN AND CERULOPLASMIN SYNTHESIS IN LIVER TISSUE. (Rus.) Volkova, Z. I. (Inst. Rheumatism, Acad. Medical Sciences USSR, Moscow, USSR). *Vopr Med Khim* 22(2): 171-176; 1976.

The effect of hepatotoxic agents (carbon tetrachloride, CCl_4, and dimethylnitrosamine, DMNA) on the rate of haptoglobin and ceruloplasmin synthesis was studied in rabbit liver tissue. Thirty-three animals received a single 1-ml/kg dose of 40% CCl_4, sc, and six animals received a single 10-mg/kg dose DMNA, iv. A marked decrease in haptoglobin content in the blood serum was noted within 24 hr after CCl_4 administration. The maximum decrease took place 2-3 days after administration, concurrent with dystrophic changes and necrosis of a significant number of parenchymal cells in the central zone of the liver. The haptoglobin content returned to normal 4-5 days after administration, simultaneous with regeneration of the parenchyma. Ceruloplasmin content in the blood serum increased as haptoglobin content decreased, and its highest level corresponded to the minimum haptoglobin level. Experiments with DMNA administration confirmed the close connection between changes in haptoglobin concentration in the blood serum and the degree of impairment of the parenchymal cells in the central zone. They indicated that haptoglobin biosynthesis takes place primarily in this zone, whereas ceruloplasmin is synthesized in the parenchymal cells of the peripheral zone. (24 references)

4336 INCREASED α_1-FETOPROTEIN PRODUCTION IN RAT LIVER INJURIES INDUCED BY VARIOUS HEPATOTOXINS. (Eng.) Watanabe, A. (Okayama Univ. Medical Sch., Shikata-cho 2-5-1, Okayama 700, Japan); Miyazaki, M.; Taketa, K. *Gann* 67(2): 279-287; 1976.

The serum α_1-fetoprotein (AFP) concentration in 5-wk-old male Sprague-Dawley rats was measured by radioimmunoassay after a single administration (ip or intragastric) of various hepatotoxins. Four days after treatment with carbon tetrachloride (0.5 ml of 20% soln/100 mg body wt), thioacetamide (20 mg), D-galactosamine (150 mg), allyl alcohol (5 mg), allyl formate (0.4 ml of 2% soln), and ethionine (100 mg), serum AFP levels were markedly elevated. The only biological parameter that correlated with the increased production of AFP was the activity of liver glucose-6-phosphate dehydrogenase (G-6-PD). However, the differences in the time courses of these two events suggest that they were caused by closely related but different mechanisms. Pretreatment of CCl_4-injured rats with N,N'-diphenyl-p-phenylenediamine or aminoacetonitrile was effective not only in lowering the increased levels of serum AFP and liver G-6-PD but also in preventing CCl_4-induced necrosis and steatosis. Treatment with a lower dose of thioacetamide (5 mg/100 g) resulted in little elevation of serum AFP and liver G-6-PD, with a markedly increased incorporation of ^{3}H-thymidine into liver DNA without any evidence of liver injury. Ethionine, which caused little necrosis of liver cells, increased both serum AFP and liver G-6-PD levels with an only small increase of hepatic DNA synthesis compared to that following thioacetamide and CCl_4. These results suggest that the elevation of serum AFP is not directly related to the stimulation of hepatic DNA synthesis. Some additional mechanisms of specific gene amplification for AFP (with respect to hepatic injury) appear to play a major role in the increased AFP production. (34 references)

4337 INVESTIGATION OF THE INFLUENCE OF COPPER AND COBALT IONS ON DIETHYLNITROSAMINE (DENA) CARCINOGENESIS IN WISTAR RATS. (Ger.) Zeller, W. J. (Institut fuer experimentalle Toxikologie und Chemotherapie, Deutsches Krebsforschungszentrum, D-6900 Heidelberg 1, Im Neueunheimer Feld 280, West Germany). *Arch Geschwulstforsch* 45(7): 634-636; 1975.

The effect of heavy metals, especially copper, on diethylnitrosamine (DENA) carcinogenesis is reported. Wistar rats of both sexes received 5 mg/kg DENA sc (back skin) weekly for a total of 215 mg/kg. One group also received 0.5 mg/kg $CuSO_4$ and another group 0.5 mg/kg $CoCl_2$, for a total dose of 21.5 mg/kg each. The metal solutions were administered with the DENA. The DENA group (24 rats) developed 10 neuroepitheliomas and 8 poorly differentiated carcinomas of the nasal cavity. The DENA-$CuSO_4$ group (24 rats) developed eight neuroepitheliomas and seven poorly differentiated carcinomas of the nasal cavity. The DENA-$CoCl_2$ group (24 rats) replicated the yield from the $CuSO_4$ group, plus one squamous carcinoma of the nasal cavity. There were six hepatomas in the DENA group, nine in the $CuSO_4$ group, and five in the $CoCl_2$ group. A hemangioendothelioma occurred in both the DENA and $CoCl_2$ groups. Simultaneous liver and respiratory tumors developed in 20/72 rats. In each case the tumor expanded from the nasal cavity through the lamina cribrosa into the olfactory bulb and to the frontal lobe. No tracheal or pulmonary growths were found. Time-to-tumor varied between 390 and 460 days. Neither copper nor cobalt was cocarcinogenic or prophylactic. (17 references)

4338 DIFFERENT SUSCEPTIBILITIES OF THE URINARY BLADDER EPITHELIUM OF ANIMAL SPECIES TO THREE NITROSO COMPOUNDS. (Eng.) Hirose, M. (Nagoya City Univ. Medical Sch., 1 Kawasumi, Mizuho-cho, Mizuho-ku, Nagoya 467, Japan); Fukushima, S.; Hananouchi, M.; Shirai, T.; Ogiso, T.; Takahashi, M.; Ito, N. *Gann* 67(2): 175-189; 1976.

Differences in the susceptibilities of the urinary bladder epithelium of Wistar rats, ICR mice, Syrian golden hamsters, and Hartley guinea pigs to three N-nitroso compounds, N-butyl-N-(4-hydroxybutyl)-nitrosamine, N-ethyl-N-(4-hydroxybutyl)nitrosamine (EHBN), and N-butyl-N-(3-carboxypropyl)nitrosamine, were examined histologically. The urinary bladder epithelium of rats was the most susceptible to all three compounds, especially to EHBN; bladder cancers developed in all rats given this compound. The bladder epithelium of mice was less susceptible than that of rats. Although the incidence of cancer was lower than in rats, that of cancer with invasion was higher. Hamsters were far less susceptible than mice; cancer developed in only 3 of 41 animals given EHBN. Guinea pigs were the least susceptible of the four species and no tumors were found. Histologically, most of the tumors induced in rats and mice were transitional cell carcinomas. The incidence of undifferentiated carcinoma was higher in mice than in rats, and cellular or structural atypism of the cancer was also greater in mice. Cancers induced in hamsters were all transitional cell carcinomas showing invasion. (27 references)

4339 AIR-WATER AND ETHER-WATER DISTRIBUTION OF N-NITROSO COMPOUNDS: IMPLICATIONS FOR LABORATORY SAFETY, ANALYTIC METHODOLOGY, AND CARCINOGENICITY FOR THE RAT ESOPHAGUS, NOSE, AND LIVER. (Eng.) Mirvish, S. S. (Univ. Nebraska Medical Center, Omaha, NB 68105); Issenberg, P.; Sornson, H. C. *J Natl Cancer Inst* 56(6): 1125-1129; 1976.

The air-water distribution ratio K_1, ether-water distribution ratio K_2, and solubility in water were measured for 17 nitrosamines, 3 nitrosamides, and 1 nitrosocyanamide. For K_1, air was analyzed either by UV absorption of ethanol extracts or by gas chromatography. K_1 at 37 C varied from less than 2×10^{-6} to 4.5×10^{-3}, with 11 compounds showing K_1 greater than 10^{-4}. A literature analysis provided data on the carcinogenicity of these N-nitroso (NNO) compounds toward the esophagus, nose, and liver of the rat. This compilation of data on physical properties and carcinogenicity of NNO compounds was then analyzed in terms of: (a) the safety of the workers handling either solutions of volatile NNO compounds or animals treated with these compounds, (b) analytic methodology, and (c) possible correlations between K_1-, K_2,- and solubility-characteristics, and carcinogenicity. Overall correlations were not observed. However, within each of five chemical groups, K_1 and K_2 tended to be associated positively with esophageal and nasal carcinogenicity and negatively with hepatic carcinogenicity. Water solubility showed the opposite associations. (21 references)

4340 IMMUNOHISTOLOGICAL STUDIES ON α_1-FETOPROTEIN AND α_1-ACID GLYCOPROTEIN DURING AZO DYE HEPATOCARCINOGENESIS IN RATS. (Eng.) Onda, H. (Faculty Medicine, Univ. Tokyo, Tokyo, Japan). *Gann* 67(2): 253-262; 1976.

The relationship in male Donryu rats between morphological changes in the liver during azo dye hepatocarcinogenesis and the concentration of cell proteins, especially α_1-fetoprotein and α_1-acid glycoprotein, was investigated by immunofluorescence. Isoelectric focusing was performed to isolate α_1-acid glycoprotein from normal rat plasma and to obtain a crude antigen containing α_1-fetoprotein from rat amnionic fluid. The specific fluorescence of α_1-fetoprotein was detected in the cytoplasm of both transitional and cancer cells, with rather a small ratio of cytoplasm per nucleus. An immunohistological study of α_1-acid glycoprotein suggests that the intracellular concentration of the protein decreases in the cytoplasm of hepatocytes in hyperplastic nodules, but increases in the cytoplasm of bile duct, oval, transitional, and cancer cells. These results indicate that hepatic cancer cells more closely resemble oval, transitional, or renewed bile duct cells than the hepatocytes in hyperplastic nodules, casting doubt on the assumption that cancer cells originate from these hepatocytes. (28 references)

4341 STUDIES ON CARCINOGENIC AZO DYES. V. THE NIH SHIFT DURING THE ARYL HYDROXYLATION OF 3'-METHYL-4-(DIMETHYLAMINO)AZOBENZENE AND 3-METHYLACETANILIDE BY THE RAT. (Eng.) Mori, Y. (Gifu Coll. Pharmacy, Mitahora higashi 5-6-1, Gifu, Japan); Toyoshi, K.; Baba, S. *Chem Pharm Bull* 24(5): 865-870; 1976.

The NIH shift during the aryl hydroxylations of deuterated or tritiated 3'-methyl-4-(dimethylamino)-azobenzene (3'-Me-DAB) and 3-methylacetanilide by a rat in vivo or in vitro has been investigated. 3'-Methyl-4'-hydroxy-4-(dimethylamino)azobenzene (3'-Me-4'-OH-DAB) was excreted in the bile and 3-methyl-4-hydroxyacetanilide in urine when 3'-Me-DAB[4'-^{2}H] was administered orally and 3-methylacetanilide[4-^{2}H, 4-^{3}H, or 5-^{2}H] was administered ip. In in vitro experiments, 3'-Me-DAB[4'-^{2}H or 4'-^{3}H] was incubated at 37 C in aerobic conditions for 1 hr with liver homogenates and 3-methylacetanilide[4-^{2}H, 4-^{3}H, or 5-^{2}H] with liver microsomes. Retention of heavy hydrogen in the 4'- or 4-hydroxylated metabolite was determined by mass spectrometry or radioactivity counting. The NIH shift was observed also during these hydroxylations. In in vitro hydroxylation of 4'-labeled 3'-Me-DAB, 94% of the tritium and 44% of the deuterium was retained. Therefore, the isotope effect in retention was great. In the case of 3-methylacetanilide, this effect was not observed, and 19% to 23% of the isotopic hydrogen was retained in the 4-hydroxylated metabolite in vivo or in vitro. Isotopic hydrogens at the position adjacent to hydroxylation in both substrates were stable. It was demonstrated that 3-methylacetanilide belongs to a class I substrate and 3'-Me-DAB to class II because of the effect of substituents on the retention of isotopic hydrogen. (13 references)

4342 HEPATIC METABOLISM OF N-HYDROXY-N-METHYL-4-AMINOAZOBENZENE AND OTHER N-HYDROXY ARYLAMINES TO REACTIVE SULFURIC ACID ESTERS. (Eng.) Kadlubar, F. F. (McArdle Lab. Cancer Res., Univ. Wisconsin Medical Center, Madison, WI 53706); Miller, J. A.; Miller, E. C. *Cancer Res* 36(7): 2350-2359; 1976.

The 3'-phosphoadenosine 5'-phosphosulfate (PAPS)-dependent esterification of N-hydroxy-N-methyl-4-aminoazobenzene (N-HO-MAB), a metabolite of the hepatocarcinogen N-methyl-4-aminoazobenzene (MAB), and several other carcinogenic and noncarcinogenic N-hydroxyarylamines are reported. Hepatic N-HO-MAB sulfotransferase activities were in the order: male rat > female rat, male rabbit, male guinea pig, male mouse > male hamster. Male rat kidney and small intestine cytosols had low activities; the other tissues studied had little or no activity. Hepatic sulfotransferase activities for N-HO-MAB and N-hydroxy-N-acetyl-2-aminofluorene displayed different pH optima and inhibitor and activator responses. The rates of the PAPS-dependent rat liver cytosol-catalyzed esterification of N-hydroxy-N-ethyl-4-aminoazobenzene, N-hydroxy-4-aminoazobenzene and N-hydroxy-1- and 2-naphthylamine were 20% to 50% of that for N-HO-MAB. Activities for trans-N-hydroxy-4-aminostilbene, N-hydroxy-2-aminofluorene, N-hydroxyaniline, and N-hydroxy-N-methyl-N-benzylamine were not detected. No microsomal NADH-dependent reduction or NADPH-dependent oxidation or cytosolic transferase reactions for N-HO-MAB, except the PAPS-dependent reaction, were detected in rat liver. (78 references)

4343 GUANINE-SPECIFIC DNA REPAIR AFTER TREATMENT OF MOUSE SKIN CELLS WITH N-METHYL-N'-NITRO-N-NITROSOGUANIDINE. (Eng.) Hennings, H. (Natl. Cancer Inst., Natl. Insts. Health, Bethesda, MD 20014); Michael, D. *Cancer Res* 36(7): 2321-2325; 1976.

The incorporation of (^{3}H)-thymidine and (^{3}H)-deoxyguanosine into DNA was studied during DNA repair in mouse skin cells treated with the skin tumor initiator N-methyl-N'-nitro-N-nitrosoguanidine (MNNG). At high, toxic levels of MNNG (25 to 50 μg/ml), repair (incorporation of the precursor into DNA that had not replicated) was demonstrated with both precursors. At lower, less toxic doses of the carcinogen (5 μg/ml), repair could not be demonstrated with (^{3}H)-thymidine, but it was clearly demonstrable with (^{3}H)-deoxyguanosine. Thus, we are apparently observing two kinds of DNA repair, one in which a single base (in this case, guanine) replaces a base lost by chemical or enzymatic depurination and the second in which more than one base is replaced, indicating synthesis of longer stretches of DNA after extensive enzymatic excision. The guanine-specific repair shown at relatively nontoxic dose levels of MNNG may be more relevant to the survival of cells than the repair demonstrated with (^{3}H)-thymidine at higher doses. (16 references)

4344 THE INDUCTION OF METASTASES IN A CHEMICALLY INDUCED CARCINOMA IN RATS (Meeting Abstract). (Eng.) Falk, R. E. (Dept. Surgery, Univ. Toronto, Toronto, Canada); Samuel, S.; MacGregor, A. B. *Clin Res* 23(5): 630A; 1975.

4345 ORGAN-SPECIFIC METABOLISM OF CARCINOGENS TO MUTAGENS (Meeting Abstract). (Eng.) Hutton, J. J. (Dept. Medicine, Univ. Kentucky, Lexington, KY 40506). *Clin Res* 24(3): 376A; 1976.

4346 MALIGNANT TRANSFORMATION OF LIVER ASSOCIATED WITH A SUSTAINED INCREASE IN CYCLIC AMP (Meeting Abstract). (Eng.) DeRubertis, F. R. (Dept. Medicine, Veterans Admin. Hosp., Pittsburgh, PA); Craven, P. A. *Clin Res* 24(3): 375a; 1976.

4347 ENHANCEMENT OF PANCREATIC TUMORIGENESIS OF 4-HYDROXYAMINOQUINOLINE-1-OXIDE (4-HAQO) BY ETHIONINE IN RATS (Meeting Abstract). (Eng.) Konishi, Y. (Dept. Oncological Pathology, Cancer Center, Nara Medical Coll., Kashihara, Nara, Japan); Denda, A.; Kawabata, H.; Sunagawa, M.; Kondo, H. *Proc Am Assoc Cancer Res* 17: 198; 1976.

4348 MALONALDEHYDE, A MUTAGENIC AGENT RESULTING FROM LIPID PEROXIDATION (Meeting Abstract). (Eng.) Goldstein, B. D. (New York Univ. Medical Center, New York, NY 10003); Mukai, F. H.; Hamburger, S. J.; McDonagh, E. M. *Clin Res* 23(5): 595A; 1975.

4349 CYTOPLASMIC DNA POLYMERASE FROM RAT LIVER AND THE EFFECTS OF CARCINOGEN TREATMENT (Meeting Abstract). (Eng.) Salisbury, J. G. (Paterson Lab., Christie Hosp., Manchester, England); O'Connor, P. J. *Br J Cancer* 34(3): 311; 1976.

4350 INCREASED CUTANEOUS CARCINOGENIC INDUCTION WITH ULTRAVIOLET RADIATION AND IMMUNOSUPPRESSIVE AGENTS (Meeting Abstract). (Eng.) Koranda, F. C. (Div. Dermatology, Univ. Colorado Medical Center, Denver, CO); Loeffler, R. T.; Koranda, D. M.; Penn, I. *Clin Res* 24(3): 494A; 1976.

4351 IN VIVO METABOLISM OF THE COLON CARCINOGEN 1,2-DIMETHYLHYDRAZINE (DMH) AND THE EFFECTS OF DISULFIRAM (Meeting Abstract). (Eng.) Fiala, E. S. (Naylor Dana Inst., American Health Foundation, Valhalla, NY 10595); Weisburger, J. H. *Proc Am Assoc Cancer Res* 17: 58; 1976.

4352 REMOVAL OF ACETYLAMINOFLUORENE ADDUCTS AND PYRIMIDINE DIMERS FROM DNA IN CULTURED MAMMALIAN CELLS (Meeting Abstract). (Eng.) Amacher, D. E. (Natl. Inst. Environmental Health Sciences, Research Triangle Park, NC 27709); Elliott, J. A. *Proc Am Assoc Cancer Res* 17: 60; 1976.

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4360 CERVICAL CANCER AND CONTRACEPTIVE PILL USE -- A CASE CONTROL STUDY (Meeting Abstract). (Eng.) Choi, N. W. (Univ. Manitoba, Winnipeg, Manitoba R3E 0W3, Canada); Nelson, N. A.; Abu-Zeid, H. A. *Am J Epidemiol* 104(3): 317-318; 1976.

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4365 EFFECT OF STEROID CONTRACEPTIVE ON CARCINOGENESIS OF THE CERVIX (Meeting Abstract). (Eng.) Stern, E. (Div. Epidemiology, Dept. Bio-Mathematics, Univ. California, Los Angeles, CA 90024); Forsythe, A.; Smedberg, P.; Youkeles, L.; Coffelt, C. *Am J Epidemiol* 104(3): 339; 1976.

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4375 INDUCTION AND ISOLATION OF ANCHORAGE-INDEPENDENT HUMAN DIPLOID CELLS AFTER SINGLE-STEP MUTAGENESIS IN VITRO (Meeting Abstract). (Eng.) Freedman, V. H. (Dept. Genetics, Albert Einstein Coll. Medicine, Bronx, NY 10461); Shin, S. *J Cell Biol* 70(2/Part 2): 310a; 1976.

4376 CYTOGENETIC EFFECTS OF OXIDANT AIR POLLUTION AND OXIDES OF NITROGEN: PRELIMINARY RESULTS FROM ENVIRONMENTAL PROTECTION AGENCY STUDIES (Meeting Abstract). (Eng.) Stebbings, J. H. (Health Effects Res. Lab., Environmental Protection Agency, Research Triangle Park, NC 27111). *Am J Epidemiol* 104(3): 360; 1976.

4377 CHRONIC LESIONS IN RATS TREATED WITH CRUDE EXTRACTS OF *FUSARIUM POAE* AND *F. SPOROTRICHIOIDES*. THE ROLE OF MOULDY FOOD IN THE INCIDENCE OF OESOPHAGEAL, MAMMARY AND CERTAIN OTHER ABNORMALITIES AND TUMOURS IN LIVESTOCK AND MAN (Meeting Abstract). (Eng.) Schoental, R. (Royal Veterinary Coll., London, England); Joffe, A. Z.; Yagen, B. *Br J Cancer* 34(3): 310; 1976.

See also:

* (Rev): 4201, 4202, 4203, 4204, 4205, 4206, 4207, 4208, 4209, 4210, 4211, 4212, 4213, 4215, 4216, 4224, 4245, 4247, 4255, 4268, 4270, 4276, 4277, 4278, 4279, 4280, 4281, 4283, 4284, 4287, 4291
* (Phys): 4383, 4404, 4405, 4406, 4407, 4424
* (Immun): 4590, 4595
* (Path): 4641, 4643, 4656, 4659, 4668, 4676
* (Epid-Biom): 4712, 4713, 4718, 4724, 4725

4378 PATHOLOGICAL ANATOMY, ETIOLOGY AND PATHOGENESIS OF REMOTE AFTEREFFECTS OF EXPOSURE TO RADIOACTIVE DUST.

(Rus.) Likhachev, Iu. P. (Scientific Res. Inst. Industrial Hygiene and Occupational Diseases, Acad. Medical Sciences USSR, Moscow, USSR). *Arkh Patol* 38(4): 18-26; 1976.

The consequences of intratracheally administered and chronically inhaled thorium (Th) and other radioactive aerosols with a specific activity from 1 x 10^{-11} to 1 x 10^{-6} Ci/g were studied in experiments with 3,000 white rats. The lungs of animals receiving promethium chloride were also investigated to determine the effect of the specific activity of α-particles. The histologic type of pulmonary carcinoma induced by Th-containing aerosols was dependent upon the intensity, total dose, and microdistribution of the radioactive dust. A dose of 150 rads accumulating in the lungs over a period of 15-21 mo increased the number of cases of reticulosarcoma, while a dose of 300 rads produced no such effects. Doses as large as 1,000-2,700 rads brought about multicentrical adenomas and glandular cancerous tumors against a background of pneumocirrhosis, but no reticulosarcomas of the lungs were noted. Adenocarcinomas and squamous cell carcinomas were distributed most often in the area in which the radioactive dust accumulated. (30 references)

4379 A PROSPECTIVE SURVEY ON THE INCIDENCE OF CHEST MALIGNANCIES AFTER REPEATED FLUOROSCOPY DURING ARTIFICIAL PNEUMOTHORAX THERAPY FOR PULMONARY TUBERCULOSIS.

(Jpn.) Kitabatake, T. (Dept. Radiology, Niigata Univ. Sch. Medicine, Niigata, Japan); Kurokawa, S.; Yamasaki, M.; Sato, T.; Kurokawa,- H.; Sakai, K. *Nippon Acta Radiol* 35(10): 895-899; 1975.

Patients with pulmomary tuberculosis who had been treated in four sanatoria in Niigata Prefecture between 1941 and 1961 were followed up with a questionnaire distributed by mail. Of 2,756 patients, 1,193 responded with complete information: 568 had been treated by artificial pneumothorax and 552 had not and were considered controls. There were 65 deaths in the pneumothorax group, none from chest malignancies; there were 40 deaths in the control group with 4 from chest malignancies. There was no evidence of increased frequency of chest malignancy, including leukemia, after pneumothorax fluoroscopy. (20 references)

4380 SEARCH FOR THE LOWEST IRRADIATION DOSE FROM THE LITERATURE ON RADIATION-INDUCED CANCER OF THE UTERUS.

(Jpn.) Yoshizawa, Y. (Dept. Radiological Health, Faculty Medicine, Univ. Tokyo, Tokyo, Japan); Kusama, T. *Nippon Acta Radiol* 35(10): 900-907; 1975.

A total of 548 previously reported cases (since 1912) of radiation-induced cancer of the uterus were surveyed to determine the lowest causative dose. All had received radiation for the treatment of the following nonmalignant gynecological conditions: functional bleeding, endometrial hyperplasia, myoma, endometritis, and/or polyps. The lowest irradiation dose was estimated at 1,000 to 1,450 rads for external x-ray irradiation and 100mg/hr for intrauterine radium therapy, corresponding of 100-1,000 rads. There were more cases of cancer of the body of the uterus than cervical cancer. The histological classifications of the induced cancers were carcinoma, sarcoma, and mixed mesodermal type. The latent period exhibited a range of 1-40 yr with an average of 10.1 yr. (105 references)

4381 INHERITED DNA REPAIR DEFECTS IN *H. SAPIENS:* THEIR RELATION TO UV-ASSOCIATED PROCESSES IN XERODERMA PIGMENTOSUM.

(Eng.) Robbins, J. H.; Kraemer, K. H.; Andrews, A. D. *In: Biology of Radiation Carcinogenesis.* Edited by J. M. Yuhas; R. W. Tennant; J. D. Regan. (New York: Raven Press); pp. 115-127; 1976.

Studies were presented and reviewed that related the different inherited DNA repair defects of xeroderma pigmentosum (XP) to several UV-associated processes. The findings suggest that the degree of impairment in unscheduled DNA synthesis does not accurately reflect the severity of the functional repair capacity defect. The finding that postreplication repair is markedly defective in the XP variants, and to a lesser degree in most of the XP groups deficient in excision repair, led to the proposition that defects in DNA repair may be causally related to the clinical signs, including skin cancer, of all known genetic forms of XP. Two models, both of which involve the somatic mutation theory of carcinogenesis, were proposed to account for the relationship between DNA repair defects and the other clinical signs of XP. One proposes that there are certain gene products common to both excision repair and postreplication repair systems and other products unique to one system or the other. The other proposes that postreplication repair is an error-prone system even in normal cells and that either a defect that enhances the error-making potential of the system itself or a defect that increases the number of lesions presented to the system would result in more errors (hence more mutations) than in normal cells. (34 references)

4382 ACTIVATION OF RNA BIOSYNTHESIS IN THE LIVER AND SPLEEN OF IRRADIATED RATS.

(Rus.) Tokarskaia, V. I. (Inst. Biological Physics, Acad. Sciences USSR, Pushchino, Moscow Oblast, USSR); Umanskii, S. R.; Skotnikova, O. I.; Kuzin, A. M. *Dokl Akad Nauk SSSR* 227(4): 988-990; 1976.

Early stages of the radiation effect on RNA biosynthesis were studied in the spleen and liver of Wistar rats subjected to ^{137}Cs γ-rays at 800 R and 409 rads/min. In the liver of irradiated rats, the specific activity of nuclear RNA, based on ^{14}C-orotic acid incorporation, increased by 2.3 times in the first 30 min. RNA activity was 50% greater than in controls after 1 hr but was similar to controls after 2 hr. The specific activity of hepatic cytoplasmic RNA was also greater than in controls, reaching a maximum after 60 min. Similar data were obtained for spleen nuclear fractions. The specific activity of the acid-soluble fraction was similar to controls in the spleen and 20% greater than controls in the liver. The increase in the specific activity of nuclear RNA was related to the activation of RNA synthesis and not to an increase in the specific activity of RNA precursors. Ultracentrifugation in a sucrose gradient showed an overproduction of large nuclear RNA 30 min after irradiation. Although the RNA spectrum after 2 hr, when repair had been completed, did not differ from controls, the irradiated organism contained residual amounts of nuclear RNA that could disturb normal cell metabolism. The increase in RNA synthesis in the liver and its inhibition in the spleen in later periods after irradiation may be secondary effects related to increased blood levels of corticosteroids.

4383 PEDIGREE ANALYSIS OF IRRADIATED HEPATOMA CELLS (Meeting Abstract). (Eng.) Sakka, M. (Tohoku Univ., Sendai, Japan); Katsuta, H.; Takaoka, T. *J Radiat Res (Tokyo)* 15(1): 54; 1974.

4384 CHROMOSOME ABERRATIONS OF MOUSE LYMPHOCYTES FOLLOWING PROTRACTED IRRADIATION (Meeting Abstract). (Eng.) Stroud, A. N. (Mammalian Biology Group, Los Alamos Scientific Lab., Los Alamos, NM 87545); Spalding, J. F.; Carpenter, S. G. *Radiat Res* 67(3): 588; 1976.

4385 THE SYNERGISTIC EFFECT OF PITUITARY ISOGRAFTS ON RADIATION INDUCTION OF HARDERIAN GLAND TUMORS (Meeting Abstract). (Eng.) Fry, R. J. (Div. Biological and Medical Res., Argonne Natl. Lab., Argonne, IL 60439); Allen, K. H.; Lombard, L. S.; Sallese, A. R.; Staffeldt, E. F.; Rust, J. H.; Ainsworth, E. J. *Radiat Res* 67(3): 570; 1976.

4386 CHANGE YOUR PATTERN BY PUTTING ON AIRS: *TETRAHYMENA PYRIFORMIS* POLYRIBOSOMES (Meeting Abstract). (Eng.) Yatvin, M. B. (Radiobiology Res. Lab., Univ. Wisconsin Medical Sch., Madison, WI 53706); Kintner, C. R.; Elson, C. E. *Radiat Res* 67(3): 573; 1976.

4387 THE EFFECT OF γ-IRRADIATION ON DEOXYRIBONUCLEOPROTEIN AS A TEMPLATE FOR RNA SYNTHESIS (Meeting Abstract). (Eng.) Hayashi, M. (Hokkaido Univ., Sapporo, Japan); Kobayashi, T.; Yoshii, . *J Radiat Res* 17(1): 9; 1976.

388 THE COMBINED EFFECTS OF HEAT AND IRRADIATION ON TRANSPLANTABILITY OF EXPERIMENTAL UMORS (Meeting Abstract). (Eng.) Fujiwara, K. (Tokushima Univ., Tokushima, Japan); Watanabe, N.; Kawa- ura, F. *J Radiat Res (Tokyo)* 17(1): 54; 1976.

389 THE EFFECTS OF PRENATAL EXPOSURE TO DIAGNOSTIC X-RAYS: ARE THEY REAL? (Meeting Abstract). (Eng.) Oppenheim, B. E. (Univ. Chicago, 50 East 59th St., Chicago, IL 60637); Griem, M. L.; eier, P. *Health Phys* 29(6): 895; 1975.

4390 EXPERIMENTAL INDUCTION OF CARCINOMA BY X-IRRADIATION IN ICR MALE MICE (Meeting Abstract). (Eng.) Fukazawa, K. (Hiroshima Univ., Hiroshima, Japan); Watanabe, H.; Kawakami, M.; Hirose, F. *J Radiat Res (Tokyo)* 15(1): 66; 1974.

4391 RADIATION-INDUCED ILEUS OF THE MOUSE SMALL INTESTINE FOLLOWING ABDOMINAL IRRADIATION. (Meeting Abstract). (Eng.) Tsubouchi, S. (Aichi Cancer Center, Nagoya, Japan); Matsuzawa, T. *J Radiat Res (Tokyo)* 15(1): 66; 1974.

4392 LATE EFFECTS OF RADIATION ON THE RAT RECTUM (Meeting Abstract). (Eng.) Black, W. C. (Cancer Res. Treatment Center, Univ. New Mexico, Albuquerque, NM 87131); Gomez, L. S.; Kligerman, M. M. *Radiat Res* 67(3): 541; 1976.

4393 ADENOSINE 3',5'-CYCLIC MONOPHOSPHATE DEPENDENT AND INDEPENDENT PROTEIN KINASE ACTIVITIES IN RAT SMALL BOWEL ADENOCARCINOMA (Meeting Abstract). (Eng.) Stevens, R. H. (Radiation Res. Lab., Univ. Iowa, Iowa City, IA 52242); Smith, D. D.; Oberley, L. W.; Shaner, J. *Radiat Res* 67(3): 569; 1976.

4394 LEVELS OF ADENOSINE 3',5'-CYCLIC MONOPHOSPHATE, PHOSPHODIESTERASE AND ADENYLATE CYCLASE IN RAT SMALL BOWEL ADENOCARCINOMA (Meeting Abstract). (Eng.) Smith, D. D. (Radiation Res. Lab., Univ. Iowa, Iowa City, IA 52242); Stevens, R. H.; Osborne, J. W.; Oberley, L. W. *Radiat Res* 67(3): 569; 1976.

4395 QUANTITATION OF AN ONCOFETAL PROTEIN(S) EXISTING IN RAT SMALL BOWEL ADENOCARCINOMA (Meeting Abstract). (Eng.) Hoffman, K. L. (Radiation Res. Lab., Univ. Iowa, 14 Medical Lab., Iowa City, IA 52242); Stevens, R. H.; Osborne, J. W.; Englund, C. W.; Cheng, H. F. *Radiat Res* 67(3): 568-569; 1976.

4396 DEMONSTRATION OF CELL-MEDIATED CYTOTOXICITY TO X-IRRADIATION INDUCED ADENOCARCINOMA OF THE SMALL BOWEL IN RATS (Meeting Abstract). (Eng.) Englund, C. W. (Radiation Res. Lab., Univ. Iowa, Iowa City, IA 52242); Stevens, R. H.; Chan, S. W.; Osborne, J. W. *Radiat Res* 67(3): 568; 1976.

4397 RADIATION INDUCED CHANGE IN CELL SURFACE CHARGE OF RAT ERYTHROCYTES AND ITS MODIFICATION BY ADDED ATP, COLCHICINE OR CYTOCHALASIN B (Meeting Abstract). (Eng.) Sato, C. (Aichi Cancer Center Inst., Nagoya, Japan); Kojima, K.; Nishizawa, K. *J Radiat Res (Tokyo)* 17(1): 55; 1976.

4398 INDUCTION OF DNA POLYMERASE DURING LYMPHOCYTE TRANSFORMATION: LACK OF INHIBITION BY X IRRADIATION (Meeting Abstract). (Eng.) Agarwal, S. S. (Inst. Cancer Res., Fox Chase Cancer Center, Philadelphia, PA 19111); Brown, D. Q.; Loeb, L. A. *J Cell Biol* 70(2/Part 2): 307a; 1976.

4399 ULTRASTRUCTURAL CHANGES IN CHROMATIN FOLLOWING X-IRRADIATION (Meeting Abstract). (Eng.) Barham, S. S. (Cellular Molecular Biology Group, Los Alamos Scientific Lab., Univ. California, Los Alamos, NM 87545); Walters, R. A.; Gurley, L. R. *J Cell Biol* 70(2/Part 2): 330a; 1976.

4400 ELECTRON MICROSCOPIC OBSERVATION ON THE DAMAGE OF CENTRAL NERVOUS SYSTEM EXPOSED TO IONIZING RADIATION (Meeting Abstract). (Eng.) Kimura, Y. (Keio Univ., Tokyo, Japan); Nagase, T.; Yamashita, H. *J Radiat Res* 17(1): 16-17; 1976.

4401 SURFACE MORPHOLOGY OF EMT6 TUMOR CELLS: SCANNING ELECTRON MICROSCOPE OBSERVATIONS OF IRRADIATED AND UNIRRADIATED CELLS IN EXPONENTIAL GROWTH AND PLATEAU PHASE (Meeting Abstract). (Eng.) Rockwell, S. (Yale Univ. Sch. Medicine, New Haven, CT 06510); Rockwell, C. *Radiat Res* 67(3): 564; 1976.

4402 PATHOLOGY IN LIFESPAN STUDY OF X-IRRADIATED FEMALE BEAGLES (Meeting Abstract). (Eng.) Chrisp, C. E. (Radiobiology Lab., Univ. California, Davis, CA 95616); Phemister, R. D.; Andersen, A. C.; Rosenblatt, L. S.; Goldman, M. *Radiat Res* 67(3): 557; 1976.

4403 COMPARATIVE CYTOGENETIC STUDIES ON RADIATION-INDUCED CHROMOSOME ABERRATIONS IN SEVERAL SPECIES OF MAMMALS (Meeting Abstract). (Eng.) Muramatsu, S. (Natl. Inst. Radiological Sciences, Chiba, Japan); Matsuoka, O. *J Radiat Res* 17(1): 29; 1976.

4404 EFFECT OF PROLACTIN IN INDUCTION AND PROGRESSION OF RAT MAMMARY TUMORS INDUCED BY RADIATION (Meeting Abstract). (Eng.) Yokoro, K. (Hiroshima Univ., Hiroshima, Japan); Ito, A.; Nakano, M. *J Radiat Res (Tokyo)* 17(1): 50; 1976.

4405 DOSE RESPONSE OF ESTROGEN INTERACTION WITH X-IRRADIATION ON THE INDUCTION OF RAT MAMMARY ADENOCARCINOMAS (Meeting Abstract). (Eng.) Stone, J. P. (Medical Dept., Brookhaven Natl. Lab., Upton, NY 11973); Holtzman, S.; Shellabarger, C. J. *Radiat Res* 67(3): 570; 1976.

4406 ANALYSIS OF DIFFERENTIAL SENSITIVITIES TO SYNCHRONIZED HELA S3 CELLS TO RADIATIONS AND CHEMICAL CARCINOGENS DURING THE CELL CYCLE (2) (Meeting Abstract). (Eng.) Watanabe, M. (Kanazawa Univ., Kanazawa, Japan); Horikawa, M. *J Radiat Res (Tokyo)* 15(1): 54; 1974.

4407 ANALYSES OF DIFFERENTIAL SENSITIVITIES OF SYNCHRONIZED HELA S3 CELLS TO RADIATIONS AND CHEMICAL CARCINOGENS DURING THE CELL CYCLE (Meeting Abstract). (Eng.) Watanabe, M. (Kanazawa Univ., Kanazawa, Japan); Horikawa, M. *J Radiat Res* 17(1): 34; 1976.

4408 MUTAGENESIS FROM ULTRAVIOLET LIGHT, X-RAYS, ^{14}C- AND ^{3}H-THYMIDINE (Meeting Abstract). (Eng.) Cleaver, J. E. (Lab. Radiobiology, Univ. California, San Francisco, CA 94113). *Radiat Res* 67(3): 589; 1976.

4409 CELL CYCLE DEPENDENT INTERACTION OF DAMAGE DUE TO IONIZING AND NONIONIZING RADIATION IN CHINESE HAMSTER CELLS (Meeting Abstract). (Eng.) Han, A. (Div. Biological and Medical Res., Argonne Natl. Lab., Argonne, IL 60439); Elkind, M. M. *Radiat Res* 67(3): 586; 1976.

4410 RADIATION-INDUCED MITOTIC DELAY IN EGGS OF A MARINE ANNELID WORM (Meeting Abstract). (Eng.) Viswanathan, G. (Div. Radiation Biology, Dept. Radiology, Case Western Reserve Univ., Cleveland, OH 44106); Rustad, R. C. *J Cell Biol* 70(2/Part 2): 177a; 1976.

4411 A MODEL FOR RADIATION DAMAGE IN MAMMALIAN CELLS BY DIRECT EFFECT AND BY INDIRECT EFFECT (Meeting Abstract). (Eng.) Michaels, H. B. (Ontario Cancer Inst., Toronto, Canada M4X 1K9); Hunt, J. W. *Radiat Res* 67(3): 582; 1976.

4412 COMPARATIVE RADIATION MUTAGENESIS, PROBLEMS AND PERSPECTIVES (Meeting Abstract). (Eng.) Abrahamson, S. (Depts. Zoology and Genetics, Univ. Wisconsin, Madison, WI 63706). *Radiat Res* 67(3): 566; 1976.

4413 QUANTIFICATION OF MAMMALIAN CELL MUTATION USING 8-AZAGUANINE RESISTANCE (Meeting Abstract). (Eng.) Jostes, R. F. (Colorado State Univ., Fort Collins, CO 80253); Hopwood, L. E.; Dewey, W. C. *Radiat Res* 67(3): 589-590; 1976.

4414 STUDIES OF THE REGENERATIVE CAPACITY OF RABBIT BONE MARROW AFTER VARIOUS DOSES OF IRRADIATION: OBSERVATIONS ON STEM CELL KINETICS (Meeting Abstract). (Eng.) Scarantino, C. W. (Univ. Rochester, Rochester, NY 14642); Rubin, P.; Gregory, P. *Radiat Res* 67(3): 590; 1976.

4415 EFFECT OF IRRADIATION ON THE ENHANCEMENT OF RNA SYNTHESIS PRIMED BY CALF THYMUS DEOXYRIBONUCLEOPROTEIN (DNP) (Meeting Abstract). (Eng.) Kobayashi, T. (Hokkaido Univ., Sapporo, Japan); Hayashi, M.; Yoshii, G. *J Radiat Res* 17(1): 9; 1976.

4416 NON-MALIGNANT LATE EFFECTS OF RADIATION AND LEUKEMIA INDUCTION (Meeting Abstract). (Eng.) Schug, W. G. (Coll. Medicine, Univ. California, Irvine, CA 92664); Ludwig, F. C. *Radiat Res* 67(3): 570-571; 1976.

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4418 STUDIES ON SELENIUM-RELATED COMPOUNDS. V. CYTOGENETIC EFFECT AND REACTIVITY WITH DNA. (Eng.) Nakamuro, K. (Dept. Hygienic Chemistry, Natl. Inst. Hygienic Sciences, Kamiyoga, Setagaya-ku, Tokyo 158, Japan); Yoshikawa, K.; Sayato, Y.; Kurata, H.; Tonomura, M.; Tonomura, A. *Mutat Res* 40(3): 177-184; 1976.

4419 ^{137}CESIUM CYCLING IN A UTAH DAIRY FARM. (Eng.) Hawthorne, H. A. (Environmental System Reviews, P.O. Box 24183, Los Angeles, CA 90024); Zellmer, S. D.; Eberhardt, L. L.; Thomas, J. M. *Health Phys* 30(6): 447-464; 1976.

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4421 RADIATION DOSIMETRY OF ^{82}SR--^{82}RB, ^{85}SR, ^{87}MSR, AND ^{90}SR-^{90}Y (Meeting Abstract). (Eng.) Bigler, R. E. (Memorial Sloan-Kettering Cancer Center, New York, NY 10021); Kostick, J. A.; Laughlin, J. S. *Radiat Res* 67(3): 558; 1976.

4422 PLUTONIUM-239 IN HUMAN ORGANS OF THE NORTHERN JAPANESE (AKITA DISTRICT) (Meeting Abstract). (Eng.) Takizawa, T. (Akita Univ., Akita, Japan); Nakamura, I.; Abe, T.; Watanabe, H.; Okabayaski, H. *J Radiat Res (Tokyo)* 17(1): 43; 1976.

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4424 β-RAY-INDUCED SKIN TUMOR FORMATION IN MICE: DISAPPEARANCE OF β-RAY-INDUCED LATENT ALTERATION (III) (Meeting Abstract). (Eng.) Hoshino, H. (Natl. Cancer Center, Tokyo, Japan); Tanooka, H. *J Radiat Res (Tokyo)* 15(1): 66; 1974.

4425 BETA RAY INDUCED SKIN TUMOR FORMATION IN MICE: EFFECT OF REPEATED EXPOSURES AND DOSE FRACTIONATION. (Meeting Abstract). (Eng.) Hoshino, H. (Natl. Cancer Center Res. Inst., Tokyo, Japan); Tanooka, H. *J Radiat Res (Tokyo)* 17(1): 51; 1976.

4426 RBE AND OER OF EXTENDED-BRAGG-PEAK HELIUM IONS: EMBRYO SURVIVAL AND TERATOGENESIS IN RATS (Meeting Abstract). (Eng.) Ward, W. F. (Dept. Radiology, Northwestern Univ. Medical Sch., Chicago, IL 60611); Aceto, H.; Sandusky, M. *Radiat Res* 67(3): 553-554; 1976.

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4428 CELLULAR LEVEL CONSIDERATIONS OF ALPHA RADIATION FROM HOT PARTICLES (Meeting Abstract). (Eng.) Todd, P. (Pennsylvania State Univ., University Park, PA 16802). *Radiat Res* 67(3): 583; 1976.

4429 DESCRIPTION OF THE HOT PARTICLE ISSUE AND CONCLUDING COMMENTS (Meeting Abstract). (Eng.) Bair, W. J. (Battelle, Pacific Northwest Lab., Richland, WA 99352). *Radiat Res* 67(3): 582-583; 1976.

4430 EXPERIMENTAL APPROACHES TO THE HOT PARTICLE ISSUE (Meeting Abstract). (Eng.) Thomas, R. G. (Mammalian Biology Group, Los Alamos Scientific Lab., Los Alamos, NM 87545). *Radiat Res* 67(3): 584; 1976.

4431 ATOMIC BOMB RADIATION STUDIES IN JAPAN. (Eng.) Finch, S. C. (Radiation Effects Res. Foundation, 5-2 Hijiyama Park, Hiroshima 730, Japan); Hamilton, H. B. *Science* 192(4242): 845; 1976.

4432 SALIVARY GLAND TUMORS IN A-BOMB SURVIVORS, IN RELATION TO A-BOMB RADIATION AND CARCINOGENESIS (Meeting Abstract). (Eng.) Takeichi, N. (Res. Inst. Nuclear Medicine and Biology, Hiroshima Univ., Hiroshima, Japan); Hirose, F.; Inoue, S.; Nishiyama, Y. *J Radiat Res (Tokyo)* 15(1): 68; 1974.

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4434 ON THE INDUCTION OF CHROMOSOMAL ABERRATIONS BY 2450 MHZ MICROWAVE RADIATION (Meeting Abstract). (Eng.) Leach, W. M. (Bureau Radiological Health, Food and Drug Administration, U. S. Dept. Health, Education and Welfare, Rockville, MD 20852). *J Cell Biol* 70(2/Part 2): 387a; 1976.

4435 CHROMOSOME ABERRATIONS BY INTERNAL DEPOSITS OF THOROTRAST (Meeting Abstract). (Eng.) Sasaki, M. S. (Tokyo Medical Dental Univ., Tokyo, Japan); Miyata, H.; Shinohara, T. *J Radiat Res (Tokyo* 17(1): 48; 1976.

4436 ESTIMATION OF ABSORBED DOSE IN THE CRITICAL ORGANS OF THOROTRAST PATIENTS (Meeting Abstract). (Eng.) Kato, Y. (Natl. Inst. Radiological Sciences, Chiba, Japan); Mori, T. *J Radiat Res (Tokyo* 17(1): 13-14; 1976.

See also:

* (Rev): 4217, 4218, 4219, 4268, 4270
* (Chem): 4314, 4316, 4350
* (Viral): 4503, 4520, 4525, 4527, 4552
* (Path): 4629, 4668
* (Epid-Biom): 4717, 4728, 4729, 4730, 4731

4437 BASE SEQUENCE COMPLEXITY OF 35S AVIAN MYELOBLASTOSIS VIRUS RNA DETERMINED BY MOLECULAR HYBRIDIZATION KINETICS. (Eng.) Baluda, M. A.; Shoyab, M.; Markham, P. D.; Evans, R. M.; Drohan, W. N. *In: Tumor Viruses. Cold Spring Harbor Symposia on Quantitative Biology.* Cold Spring Harbor Lab. (Cold Spring Harbor, NY): vol. 39, pp. 869-874; 1975.

To determine whether avian myeloblastosis virus (AMV) contains identical or different 35S subunits, the complexity of 35S RNA was analyzed by DNA-RNA hybridization. A 400- to 1,000-fold excess of ^{3}H-labeled 35S AMV RNA in solution was hybridized with complementary DNA immobilized on filters. Under these conditions, hybrid formation rate was inversely proportional to the base sequence complexity of the RNA, and the kinetic complexity of the unknown RNA molecules could be estimated by comparison with species of known complexity (chicken 18S, 28S, and 18S + 28S ribosomal RNAs, rRNAs). The hybridization reactions were carried out in 6 x SSC containing 50% formamide and in 4 x SSC at the predetermined optimal temperatures for each species (1 x SSC = 0.15 M NaCl and 0.015 M sodium citrate). By comparison with the Crt-1/2 (Cr being the molar radionucleotide concentration and t-1/2 the time in seconds to achieve 50% of maximum hybridization) of any of the ribosomal RNAs as reference, the analytical complexity of 35S AMV RNA was about 3.16×10^6 daltons in both experiments. The kinetic complexity of the three rRNA species was in close agreement with their molecular wt. When hybridization temperatures were such that the reaction rates were equally reduced to 75% of their optima, the complexity of 35S AMV RNA in 4 x SSC was 3.68×10^6 daltons. Thus, the number of unique nucleotide sequences in 35S AMV RNA is equivalent to an average complexity of 3.3×10^6 daltons, and it appears that the 35S AMV RNA molecules are either identical or very similar in base sequence complexity. (24 references)

4438 AMV RNA TRANSCRIPTION IN CELL-FREE SYSTEMS AND PROPERTIES OF IN VITRO CHROMATIN-DIRECTED RNA SYNTHESIS. (Eng.) Monroy, G.; Jacquet, M.; Groner, Y.; Hurwitz, J. *In: Tumor Viruses. Cold Spring Harbor Symposia on Quantitative Biology.* Cold Spring Harbor Lab. (Cold Spring Harbor, NY): vol. 39, pp. 1033-1041; 1975.

To elucidate the mechanism of RNA tumor virus expression and provide information on transcription in eukaryotic cells, two cell-free systems that transcribe viral RNA were developed. The first system utilized nuclei isolated from chicks infected with avian myeloblastosis virus (AMV) and the second used chromatin isolated from these nuclei and an exogenous eukaryotic RNA polymerase. Viral RNA sequences were detected by molecular hybridization with a DNA probe. Analysis of RNA synthesis in nuclei isolated from myeloblasts of AMV-infected chicks indicated that AMV RNA is transcribed from proviral DNA present in the nucleus and that some transcriptional specificity is maintained in the cell-free system. When myeloblast chromatin served as a template for synthesis catalyzed by a nucleoplasmic RNA polymerase, approximately 0.9% of the total viral RNA formed stable RNA-DNA hybrid structures with the DNA probe. Sequences in the product that formed RNA-cDNA (complement DNA) hybrid structures were present in 35S RNA. RNA synthesized by calf thymus RNA polymerase B in this system was heterogeneous in size, ranging from 5S-50S, with a broad peak at the top of the sucrose gradient. The addition of heparin during the incubation period stimulated RNA synthesis and significantly increased the proportion of large molecular wt RNA in the product. Heparin had no effect on the size distribution of the products synthesized from either single- or double-stranded DNA. The data support the hypothesis that the expression of viral information is mediated by nucleoplasmic RNA polymerase. The findings are also consistent with the suggestion that gene activation in eukaryotic organisms results from the unwinding of segments of chromatin DNA. (35 references)

4439 CYCLING OF RNA-DIRECTED DNA POLYMERASE ON NATURAL AND SYNTHETIC RNA TEMPLATES. (Eng.) Kruegar, L. J. (Natl. Heart and Lung Inst., Natl. Insts. Health, Bethesda, MD 20014); Weiss, G. B.; Merrick, W. C.; Lloyd, M. A.; Anderson, W. F. *Nature* 260(5549): 363-365; 1976.

In vitro conditions are described such that each molecule of RNA-directed DNA polymerase [from avian myeloblastosis virus (AMV)] synthesizes several to more than 100 transcripts of full-sized complementary DNA (cDNA) from viral messenger RNA (mRNA), depending on whether natural or synthetic templates are used. Eighty percent pure AMV (strain BAL) DNA polymerase was purified by collecting the virus from plasma by high speed centrifugation, batch elution from DEAE-cellulose, and chromatography on phosphocellulose. Optimal concentration of DNA polymerase was 0.24 pmol/50 µl reaction mixture. The time course of globin mRNA-directed deoxycytosine monophosphate synthesis was compared with deoxyguanosine monophosphate (dGMP) incorporation directed by poly(C). The globin mRNA-directed synthesis of cDNA remained linear for only 20 min, compared with more than 180 min for poly(C)-directed polymerization. Poly(C)-directed incorporation of dGMP yielded 29 pmol of full-sized product, and thus 115 cDNA molecules per enzyme molecule. The use of globin mRNA as template yielded 1.8 pmol of full-sized product, and hence 7.2 molecules per molecule of DNA polymerase. Whether the ability to utilize more than a single template applies in vivo or whether reverse transcriptase is intrinsically controlled to polymerize only a single viral molecule awaits future study.

4440 PHENOTYPIC MIXING BETWEEN VESICULAR STOMATITIS VIRUS AND AVIAN RNA TUMOR VIRUSES. (Eng.) Weiss, R. A.; Boettiger, D.; Love, D. N. *In: Tumor Viruses. Cold Spring Harbor Symposia on Quantitative Biology.* Cold Spring Harbor Lab. (Cold Spring Harbor, NY): vol. 39, pp. 913-918; 1975.

Reciprocal phenotypic mixing between vesicular stomatitis virus (VSV) and various strains of avian RNA tumor virus (ATV) was studied. The mutant VSV tl 17 was grown in chicken or golden pheasant cells that had been preinfected with avian leukemia or sarcoma viruses, and determinations were made of the host range of VSV(ATV) pseudotypes for chicken and rat cells, the plating efficiency of VSV(ATV) pseudotypes on avian and mammalian cells, and the effect of host resistance genes on the penetration of VSV-(ATV). The infectivity of the VSV pseudotypes in chicken and pheasant cells depended on the ATV envelope antigens and was restricted to the host range of the ATV. VSV tl 17 appeared to be complemented by ATV envelope components. The plating efficiency of the VSV(ATV) pseudotypes on mammalian cells paralleled the oncogenicity of these strains in these cells, indicating that viral penetration was a limiting factor in mammalian tropism. The penetration of VSV(RAV-0) was inhibited by the *Ie* allele, which behaves as a dominant epistatic inhibitor of susceptibility. The exclusion of virus by the *Ie* gene product was specific to subgroup E viruses, and all cells carrying the *Ie* allele appeared to be positive for chick helper factor (chf). When cells that were producing RSV were superinfected with VSV, ATV particles with envelope antigens of VSV were made. A comparison of three VSV ts mutants indicated that ts 1026 was 10 times more efficient at inducing RSV(VSV) pseudotypes than were ts 114 and tl 17; the inverse was found for VSV(ATV) pseudotypes. The results indicate that VSV pseudotypes determined by ATV are useful in distinguishing restrictions to viral penetration from restrictions to virus expression occurring later in the replication cycle. (29 references)

4441 INTRACELLULAR RESTRICTION ON THE GROWTH OF INDUCED SUBGROUP E AVIAN TYPE C VIRUSES IN CHICKEN CELLS. (Eng.) Robinson, H. L. (Stanford Univ. Sch. Medicine, Stanford, CA 94305). *J Virol* 18(3): 856-866; 1976.

Experiments were performed on the intracellular restriction of growth of RAV-60, RAV-0, and subgroup E induced leukosis viruses (ILVs) in chicken embryo fibroblasts of K28, line 15, and crosses of K28 and line 15 chicken cells. Subgroup E avian type C viruses produced by bromodeoxyuridine (BUdR)-treated 100 x 7, line 7, or line C chicken cells were restricted in their intracellular growth on K28 chicken cells. Cells from embryos of line 15 chickens bred with K28 chickens did not restrict the growth of the subgroup E ILVs. This result indicates that the phenotype for the intracellular restriction of the growth of subgroup E ILBs found in K28 cells is recessive. Long-term growth of the subgroup E ILVs in K28 cells resulted in the appearance of subgroup E virus that grew well on K28 cells. No change in growth characteristics was observed for subgroup E ILVs grown in line 15 cells, indicating that appearance of nonrestricted virus occurred only during growth of the subgroup E ILVs on a restrictive host. RAV-60, a subgroup E virus closely related to the ILVs, had the same growth characteristics as the subgroup E ILVs. RAV-60, a subgroup E virus formed by recombination of exogenous avian leukosis virus with endogenous subgroup E virus coat information, grew well on both line 15 and K28 cells. These results suggest the possibility that the phenotypes for rapid intracellular growth of E ILV and for increased production of endogenous virus after growth in BUdR reflect the expression of the same locus.

4442 IMMUNE RESPONSE TO ONCORNAVIRUSES AND TUMOR-ASSOCIATED ANTIGENS IN THE CHICKEN. (Eng.) Bauer, H. (Institut fuer Virologie, Bereich Humanmedizin, Justus Liebig-Universitaet, 6300 Giessen, Frankfurter Strasse 107, West Germany); Kuhrt, R.; Rohrschneider, L.; Gelderblom, H. *Cancer Res* 36(2): 598-602; 1976.

Two viral-directed antigens, identified on the surface of oncornavirus-infected chick embryo cells, were characterized. One is identical with the major virus type-specific envelope antigen, which is glycoprotein with a molecular wt of 85,000. The second antigen (tumor-specific cell surface antigen) is specific for transformed cells, that is, absent from productively infected but nontransformed cells. This antigen has been identified as a glycoprotein with a molecular wt of 100,000 and was not found in the mature virion. Remarkably, both antigens induce humoral as well as cellular immunity in the chicken. It could be shown that cells can be killed in cytotoxic assays via either the tumor-specific cell surface antigen or the virus envelope glycoprotein alone.

4443 INITIATION SITES OF ROUS SARCOMA VIRUS RNA-DIRECTED DNA SYNTHESIS IN VITRO. (Eng.) Cashion, L. M. (Institut fuer Molekularbiologie I, Univeristaet Zurich, 8049 Zurich, Switerland); Joho, R. H.; Planitz, M. A.; Billeter, M. A.; Weissmann, C. *Nature* 262(5565): 186-190; 1976.

The origin and complexity of the DNA synthesized in vitro by disrupted Rous sarcoma virus, Prague B strain (Pr-B RSV), were studied with hybridization techniques. The most frequent DNA species corresponded to a region that was not more than 200 nucleotides in length and included the 5' terminus of the RNA. Several other regions of the RNA were hybridized to DNA. These data suggest that the major initiation site is located near the 5' terminus, but that DNA synthesis may also originate at various other positions throughout the genome. Digestion of the hybrid DNA-RNA showed that 65% of the oligonucleotides were of the T_{13} variety. Twenty percent of the oligonucleotides were T_1, a segment from the middle section of the RNA genome. The authors discuss experiments that demonstrate that oligonucleotide T_{13} contains the capping group and thus constitutes the 5' terminus of Pr-B RSV RNA. Since the data suggest several initiation sites for DNA synthesis, various mechanisms for synthesis at the different sites are discussed. The relationship between in vitro and in vivo DNA synthesis has not been determined. (32 references)

4444 ELECTRON MICROSCOPE MEASUREMENTS OF ROUS SARCOMA VIRUS RNA. (Eng.) Delius, H.; Duesberg, P. H.; Mangel, W. F. *In: Tumor Viruses. Cold Spring Harbor Symposia on Quantitative Biology.* Cold Spring Harbor Lab. (Cold Spring Harbor, NY): vol. 39, pp. 835-843; 1975.

The subunit structures of the RNA of Rous sarcoma virus (RSV) and tobacco mosaic virus (TMV) were studied under the electron microscope. Bacteriophage T4 gene 32 protein was used to mildly denature and extend the RSV 60-70S and 30-40S RNAs so that, in conjunction with the protein monolayer spreading technique of Kleinschmidt, their structure could be visualized. With double-stranded DNA as an internal length standard, the molecular wt of the RNA-gene 32 protein complexes was determined. Bacteriophage T4 gene 32 protein differentially affected the hydrodynamic properties of the 60-70S and 30-40S RNAs; the upper limit in the length distribution, as measured with the electron microscope, could be correlated with a hydrodynamic property in each case. By this method, the molecular wt of TMV RNA was 2.2 x 10^6 daltons, a figure that agrees closely with those obtained by other methods. The 30-40S RNA, in the presence of gene 32 protein, appeared to be a linear molecule with an average molecular wt of 2.74 x 10^6 daltons (with an uncertainty of 10%). The 60-70S RNA-protein complexes generally appeared to consist of several linear molecules held together at many different points. The average length was 6.8 μm, which corresponds to a molecular wt of 6.1 x 10^6 daltons. There was also a second peak in the length distribution, between 2.8 and 4.2 μm; the molecular wt of the RNA in this size range averaged 3.1 x 10^6 daltons. Incubation of the 60-70S RNA in 8 M urea at low ionic strength resulted in a change of the sedimentation coefficient to 30-40S; no such change was observed if the incubation occurred in the presence of 0.1 M NaCl. (33 references)

4445 THE CHICK FIBROBLAST CELL SURFACE AFTER TRANSFORMATION BY ROUS SARCOMA VIRUS.

(Eng.) Robbins, P. W.; Wickus, G. W.; Branton, P. E.; Gaffney, B. J.; Hirschberg, C. B.; Fuchs, P.; Blumberg, P. M. *In: Tumor Viruses. Cold Spring Harbor Symposia on Quantitative Biology.* Cold Spring Harbor Lab. (Cold Spring Harbor, NY): vol. 39, pp. 1173-1180; 1975.

Secondary chick embryo fibroblasts transformed by the ts 68 mutant of the Schmidt-Rupin strain of Rous sarcoma virus (RSV) were used to compare lipid fluidity, cellular proteins, and actin in normal and transformed cell membranes. The transformed fibroblasts have a normal morphology at 41 C but rapidly assume a transformed morphology at 36 C. Although ts 68-infected cells at 36 and 41 C may be slightly more rigid than the corresponding normal cells, there was no difference in fluidity that could be clearly associated with transformation. Similarly, there were no consistent differences in the fluid lipid regions of normal and transformed plasma membranes. The plasma membranes of transformed cells also showed decreased levels of three specific cellular proteins: omega, delta, and zeta. Omega, a polypeptide with a molecular wt of about 206,000, ceased to be synthesized or began to degrade almost immediately on shift from 41 to 36 C. Delta, which appears to be a glycoprotein, was synthesized at control levels for the first 3 hr after the temperature shift in both infected and uninfected cells, after which the synthesis rate dropped (to approximately 10%-20% of the 41 C rate in uninfected cells). A glycoprotein of about 70,000 daltons was also present in higher amounts in infected cells at 41 C than at 36 C; this may represent an accumulation of viral protein. The zeta polypeptide is extracellular, exhibits protease sensitivity, decreases in concentration in transformed cells, and may be related to a procollagen type of molecule. Its degradation may not represent a fundamental requirement for transformed cell morphology. RSV transformation appeared to reduce the amount of ''polymerized'' actin and increase ''monomeric'' actin, but had no effect on the total quantity of actin in the plasma membrane. Alterations in actin biochemistry may be involved in the production of some of the transformed cell phenotypic properties. (13 references)

4446 TEMPERATURE-SENSITIVE CHANGES IN SURFACE MODULATING ASSEMBLIES OF FIBROBLASTS TRANSFORMED BY MUTANTS OF ROUS SARCOMA VIRUS.

(Eng.) Edelman, G. M. (Rockefeller Univ., New York, NY 10021); Yahara, I. *Proc Natl Acad Sci USA* 73(6): 2047-2051; 1976.

The hypothesis that surface modulating assemblies (SMA) containing microfilaments and microtubules are altered after cellular transformation was tested on cells infected with temperature-sensitive (ts) mutants of avian sarcoma virus. Untransformed cells (mouse 3T3 and chick fibroblasts), cells transformed by simian virus 40 (SV 3T3), and chick fibroblasts infected with Schmidt-Ruppin strain of Rous sarcoma virus (SR-RSV-A-infected cells) were first compared for differences in microfilament and microtubule patterns after treatment with fluorescein-labeled antibodies to actin and tubulin. Transformed cells showed disappearance of ordered stress microfilaments and thickened or diffuse alterations of microtubular arrays. At restrictive temperatures (41 C), chick fibroblasts infected with ts 68 of Rous sarcoma virus showed normal patterns of stress filaments and radial microtubular arrays originating in one or two centrioles. At permissive temperatures (37 C), these patterns were disordered and resembled those of SR-RSV-A-infected cells. After a shift from 41 C to 37 C, the changes in microtubules were observed in the majority of cells within 1 hr. These changes were reversible and did not result from the inability of tubulin to polymerize. In ts 68-infected cells at permissive temperatures, concanavalin A induced much less surface modulation (inhibition of receptor mobility) than at restrictive temperatures. These results suggest that cellular transformation alters both the structure and function of SMA and prompt the hypothesis that products of viral transforming genes may affect SMA with a consequent loss of growth control. (14 references)

4447 CHARACTERISTICS OF A HUMAN CELL LINE SUCCESSIVELY TRANSFORMED BY ROUS SARCOMA VIRUS AND SIMIAN VIRUS 40.

(Eng.) Kuwata, T. (Dept. Microbiology, Sch. Medicine, Chiba Univ., Chiba 280, Japan); Oda, T.; Sekiya, S.; Morinaga, N. *J Natl Cancer Inst* 56(5): 919-926; 1976.

Since human cells transformed by Rous sarcoma virus (RSV) and simian virus 40 (SV40) are difficult to subculture continuously, an attempt was made to transform human cells successively by RSV and SV40 and to establish permanent double-transformant cells.

Human embryo cells were successively transformed by the Schmidt-Ruppin (SR) strain of RSV and SV40 in vitro, and the double transformant HuE 13 RS was established. From this cell line the two clonal cell lines RSa and RSb were isolated. SV40 T antigens were demonstrated in both lines by the fluorescent antibody technique, and the presence of RSV genomes was verified in one RSb clone by focus formation after fusion with chick embryo cells. The growth of these cells was affected by dibutyryl cAMP without marked morphologic changes. Although the sensitivity of various cells to interferon preparations is not always equal, sensitivity to the anticellular action of human leukocyte interferon was one of the conspicuous characteristics of RS cells. (37 references)

4448 REMOVAL OF INHIBITORS OF PARTICULATE REVERSE TRANSCRIPTASE IN HUMAN MILK. (Eng.) Sanner, T. (Norwegian Radium Hosp., Montebello, Oslo 3, Norway). *Anal Biochem* 71(1): 304-307; 1976.

Factors that influence the reverse transcriptase activity in human milk were studied using various methods. When reverse transcriptase from B particles in the milk of the R-III mouse was assayed in the presence of the ''virion fraction'' from human milk purified by discontinuous or continuous sucrose or discontinuous glycerol density gradient centrifugation, nearly complete inhibition of the enzyme was obtained. Centrifugation of the purified ''virion fraction'' through a cushion containing 25% Metrizamide completely removed the inhibitors. Subsequent experiments with murine mammary cancer virus showed that the discontinuous sucrose or glycerol gradient could be substituted by a discontinuous Metrizamide gradient with 25% Metrizamide on the top layer and 30% Metrizamide on the bottom.

4449 ENDOGENOUS MAMMARY TUMOR VIRUSES IN MICE. (Eng.) Bentvelzen, P. *In: Tumor Viruses. Cold Spring Harbor Symposia on Quantitative Biology.* Cold Spring Harbor Lab. (Cold Spring Harbor, NY): vol. 39, pp. 1045-1049; 1975.

The existence of endogenous mammary tumor viruses (MTV) in mice was studied. In the GR mouse strain, the mammary tumor-inducing agent is transmitted by both the female and male parents. Introduction of the GR strain MTV (MTV-P) into other mouse strains (C3Hf) does not lead to male transmission of the virus, indicating a strong association between the GR genome and MTV-P with regard to gamete-borne transmission. The abnormal mode of MTV-P transmission in the GR strain is controlled by a single dominant Mendelian factor. MTV-L (a virus strain derived from mouse strains from which the milk factor has been removed by foster nursing) is also transmitted at conception by either C3Hf parent, the property apparently being controlled by a single gene located in the same linkage group as the GR gene for MTV-P. EMT6 mammary tumor cells, derived from a BALB/c tumor, were mixed with sperm from various mouse strains; only sperm from the GR strain induced the synthesis of MTV-specific antigens. Genetic transmission was also deduced based on the segregation of MTV-P and MTV-L markers in a test cross of the F_1 hybrids between C3Hf and MTV-P. The inducibility of type B oncornaviruses in mouse kidney cells by 5-bromo-2'-deoxyuridine (BUdR) was studied. Exposure to BUdR induced virus in a variety of mouse strains, but it persisted only in C3Hf, DBAf, Gr, and the low-cancer strain BALB/c. The endogenous MTV of BALB/c (MTV-O) was induced in BALB/c by lethal doses of radiation. The data suggest that different genetic systems control the expression of endogenous MTV. Some affect only that germinal provirus with which they are genetically linked, while others have an influence on other germinal proviruses as well as somatic proviruses. (39 references)

4450 LACK OF INTERFERENCE IN ANTIGEN PRODUCTION IN VITRO BETWEEN B-TYPE AND C-TYPE ONCORNAVIRUSES AS DETERMINED BY DOUBLE STAIN IMMUNOFLUORESCENCE. (Eng.) Van Pelt, F. G. (Inst. Experimental Gerontology TNO, 151 Lange Kleiweg, Rijswijk, The Netherlands); Houweling, A.; Aarssen, A. M.; Bentvelzen, P. *Eur J Cancer* 12(4): 321-323; 1976.

Cultures of mammary tumors induced in BALB/c mice by murine mammary tumor virus (MTV) were examined for the presence of antigens of MTV and of Rauscher murine leukemia virus (RLV). Double stain immunofluorescence (fluorescein and tetramethylrhodamine isothiocyanate) demonstrated that a high percentage of cells produced both types of viral antigens. There was no statistical indication of mutual interference between the two viruses; although the number of C-type particles detected by the electron microscope increased during subsequent passage of the cell line, MTV release was not inhibited. The results suggest that any in vivo interference between MTV and RLV is not due to intracellular competition between the viruses.

4451 GLUCOCORTICOID INDUCTION OF MURINF MAMMARY TUMOR VIRUS IN VITRO. (Eng.) Parks, W. P.; Scolnick, E. M.; Ransom, J. C. *In: Tumor Viruses. Cold Spring Harbor Symposia on Quantitative Biology.* Cold Spring Harbor Lab. (Cold Spring Harbor, NY): vol. 39, pp. 1151-1158; 1975.

Several cell lines developed from spontaneously occurring mammary adenocarcinomas were used to study the biological and biochemical parameters of murine mammary tumor virus (MMTV) in vitro. Immunoassays and determinations of the time course of MMTV synthesis and the inducibility of various MMT cell lines were performed after dexamethasone (1 μg/ml) treatment of the cells. In addition, the supernatant DNA polymerase activity, the dose-response of dexamethasone stimulation, and the ability of various hormones to induce MMT synthesis or interfere with dexamethasone stimulation of MMTV were measured. Addition of dexamethasone to a cell line increased supernatant polymerase activity significantly; it was first detected at 16 hr. Polymerase levels increased another 20 times over the next 72 hr, then declined slightly. The continued presence of dexamethasone was required for increased MMTV synthesis. Dexamethasone stimulation of murine cells proceeds through a highly specific mechanism to increase the levels of type B viral RNA transcription without increasing those of type C significantly. There was no evidence that dexamethasone treatment resulted in de novo transcription of MMTV from cells not already expressing constitutive amounts of MMTV gene sequences. The absolute levels of cytoplasmic receptor probably did not account for the biological patterns of MMTV induction. MMTV production was stimulated by dexamethasone in concentrations as low as 2.8×10^{-10} M (physiologic levels). MMTV synthesis was also induced by hydrocortisone, corticosterone, and aldosterone, although these hormones were not as potent as dexamethasone. Progestins, estrogens, androgens, spermidine, insulin, and prolactin were ineffective as inducers, although testosterone, androstendione, progesterone, and 17α-hydroxyprogesterone were inhibitory. It appears that dexamethasone stimulation of MMTV synthesis proceeds via a highly specific allosteric interaction with a cytoplasmic receptor. (29 references)

4452 CHARACTERISTICS OF A SURFACE-ADHERENT SUBLINE DERIVED FROM FRIEND ERYTHROLEUKEMIA CELLS IN CONTINUOUS SUSPENSION CULTURE. (Eng.) Demsey, A. (Div. Labs. and Res., New York State Dept. Health, Albany, NY 12201); Grimley, P. M. *Cancer Res* 36(2): 384-393; 1976.

A surface-adherent cell population developed spontaneously from Friend erythroleukemia cells (FLC745) which previously had grown continuously in suspension cultures. The adherent cells were transferred through more than 60 passages and apparently represent a stable variant, herein designated FLC745-Ad. The cell size, chromosome complement, and tumorigenicity in DBA/2 mice were similar to the parental FLC745 line, although the FLC745-Ad cultures grew at a slightly slower rate and synthesized less hemoglobin in response to dimethyl sulfoxide. Production of C-type particles was abundant in both suspension and adherent cells, but the latter appeared to contain more intracisternal A-type particles. Scanning electron microscopy disclosed that most adherent cells were globoid and nonspreading. They attached to the substrate by a circumferential lamellar skirt of unusual breadth. Adhesion of the FLC745-Ad cells was not influenced by serum concentration, trypsinization, 5-bromodeoxyuridine, or $N^6,O^{2'}$-dibutyryl adenosine 3':5'-monophosphate. Treatment with cytochalasin B increased the cell size and the area of cell attachment.

4453 SURFACE MORPHOLOGY OF SPLEEN CELLS FROM MICE INFECTED WITH FRIEND LEUKEMIA VIRUS. (Eng.) Farber, P. A.; Specter, S.; Friedman, H. *In: Tumor Virus Infections and Immunity*. Edited by R. L. Crowell; H. Friedman; J. E. Prier. (Baltimore: Univ. Park Press): pp. 231-239; 1976.

Alterations in the surface morphology of spleen cells from BALB/c mice infected with Friend leukemia virus (FLV) are described. Within a few days after infection, electron microscopy revealed a decrease in the number of cells covered with villi and an increase in the number of smooth cells, while cells with a few villi had increased to 40%. The number of smooth cells increased as infection progressed, so that at 10 days most of the cells had a smooth configuration and at 17 days few normal lymphocytes remained. At 30 days after infection, there were lymphocytes with numerous blebs as well as punctate lesions resembling holes. Lysed lymphocytes seemed to result from the coalescence of these pores. The results are discussed in terms of loss of B cell function and the immunosuppression that accompanies FLV infection. (17 references)

4454 *GAG* GENE OF MAMMALIAN TYPE-C RNA TUMOUR VIRUSES. (Eng.) Barbacid, M. (Lab. RNA Tumor Viruses, National Cancer Inst., Bethesda, MD 20014); Stephenson, J. R.; Aaronson, S. A. *Nature* 262(5569): 554-559; 1976.

Radioimmunoassays were used to study the components and the genetic sequence of the *gag* gene of a temperature-sensitive Rauscher murine leukemia virus (MuLV) mutant defective in precursor-polypeptide cleavage (mutant *ts*25). NIH/3T3 mouse cells were infected with *ts*25 at nonpermissive temperatures, and the various polypeptide precursors and their intermediate cleavage products were studied. Antigenic activities corresponding to four peptides (p30, p15, p12, and p10) migrated as a single precursor (Pr65) with a molecular wt of 65,000-68,000. Various polypeptides containing the individual peptides were present with molecular wt lower than 65,000. The translation products of various mammalian sarcoma virus-transformed cells were analyzed in order to sequence the type C viral *gag* gene. The data strongly suggest that p15 is the amino-terminal protein of the *gag* gene product and that p10 is the carboxy-terminal protein. The entire gene sequence is thought to be NH_2-p15-p12-p30-p10-COOH. Since the viral RNA genome is the positive strand, the genetic information in the *gag* gene would be arranged in an identical manner from the 5' to 3' end of the RNA molecule. The evidence and reasoning is presented for the hypothesis that the type C viral genome contains one protein synthesis initiation site at the 5' end and that the *gag* gene is close to the end of the RNA molecule.

4455 DNA-RNA HYBRID IN CELLS INFECTED BY MURINE LEUKEMIA VIRUS. (Eng.) Takano, T.; Hatanaka, M. *In: Tumor Viruses. Cold Spring Harbor Symposia on Quantitative Biology*. Cold Spring Harbor Lab. (Cold Spring Harbor, NY): vol. 39, pp. 1009-1014; 1975.

The hypothesis that integration of the viral genome is a prerequisite for the production of progeny virus and the transformation of cultured cells by oncogenic RNA tumor viruses was studied. Monolayers of mouse embryo fibroblasts were infected with ^3H-uridine-labeled Rauscher leukemia virus (RLV), and the nucleic acids of the infected cells were analyzed by Cs_2SO_4 density centrifugation. Radioactive RNA-DNA hybrid molecules were found: one was detected in the cytoplasmic fraction, had a molecular size of 4-6S, and had a density of 1.56 (RNA:DNA = 1:1); the other was found in the nuclear fraction, had a sedimentation rate of 65S, and had a density (1.48) close to that of the host DNA. Cytosine arabinoside inhibited the formation of cytoplasmic and nuclear hybrid molecules, but hydroxyurea inhibited only the formation of the hybrids in the nucleus. It is hypothesized that the viral RNA and viral DNA polymerases, both originating from the infected virions, synthesize RNA-DNA hybrid molecules (RNA:DNA + 1:1) in the cytoplasm. These hybrid intermediates are translocated into the cellular nucleus, whereupon further DNA synthesis occurs in the nucleus, leading to a shift in the density of the hybrids to closer to that of the host DNA. The RNA-DNA hybrid molecules of the nucleus may be the intermediates in the processes leading to integration of the viral genome into the host chromosome. (24 references)

4456 INTRANUCLEAR INCLUSIONS IN HEMATOPOIETIC CELLS DURING EARLY STAGES OF VIRAL LEUKEMOGENESIS.

(Ukr.) Butenko, Z. A. (Inst. Problems Oncology, Acad. Sciences Ukrainian SSR, Kiev, USSR); Naumenko, O. I. *Dopov Akad Nauk Ukr RSR Ser B* (3): 274-277; 1976.

Electron microscopy of the spleen medulla cells of Balb/C mice 2 days after ip infection with Rauscher leukemia virus showed intranuclear inclusions in 50% of the hematopoietic cells compared with 9%-10% in the controls. The types of nuclear bodies found included the simple (0.3-0.5 μ in diameter) or fibrous (100 A thick) bodies located near the nucleolus or in the nuclear periphery, granular (200 A in diameter) and globular bodies with fibrous components and intermediate forms, and tubular forms (300 A in diameter) surrounded by fibrous structures. The fibrous inclusions were proteins and the granular inclusions were ribonucleoproteins. Increases in Golgi apparatus, hypertrophy of the endoplasmic reticulum, and structural changes in the mitochondria were also observed. Increases in intranuclear inclusion levels in these infected cells are indicative of the activation of ribonucleoprotein synthesis in early leukemogenesis. (12 references)

4457 TEMPLATE SPECIFICITIES OF THE RNA DEPENDENT DNA POLYMERASE OF DIFFERENT MURINE C-TYPE VIRUS PARTICLES.

(Eng.) Kontor, E. J. (Christ Hosp. Inst. Medical Res., 2141 Auburn Ave., Cincinnati, OH 45219); Krueger, R. G. *Biochem Biophys Res Commun* 70(4): 1235-1242; 1976.

The crude RNA-dependent DNA polymerase (RDDP) of seven different type C viruses, [AMV, Kirsten-MSV produced by NRK or NIH/3T3 cells, Moloney-MuLV, Kirsten-MuLV, the murine myeloma-associated virus MuMAV) from FLOPC-1 and MOPC-21] in normal rat kidney cells was analyzed for its ability to utilize four different synthetic RNA/DNA hybrids or three different DNA/DNA duplexes as templates. The polymerases from AMV and murine sarcoma or leukemia viruses were distinctly different in their template-stimulated activities, and the two MuMAV polymerases were different from all of the other enzymes. MuMAV RDDPs were not stimulated by any of the synthetic RNA/DNA hybrid templates to the same level as the enzymes of the other viruses, and their ability to distinguish between templates was also different. The studies indicate that: (a) structurally distinct AMV and murine oncornavirus DNA polymerases are also enzymatically distinct; (b) immunologically closely related viral RDDP enzymes have differing template-stimulating activity; and (c) the RDDP of MuMAV particles and the polymerases of other murine oncornaviruses and AMV are distinctly different. (18 references)

4458 TRANSLATION OF MURINE LEUKEMIA VIRUS RNA IN CELL-FREE SYSTEMS FROM ANIMAL CELLS.

(Eng.) Kerr, I. M. (Dept. Biology, Massachusetts Inst. Technology, Cambridge, MA 02139); Olshevsky, U.; Lodish, H. F.; Baltimore, D. *J Virol* 18(2): 627-635; 1976.

The virion RNA of Moloney murine leukemia virus (MuLV) was translated in eukaryotic cell-free systems derived from mouse L and human HeLa cells. Gel electrophoresis showed that at least three polypeptides, approximately 60,000, 70,000, and 180,000 in apparent molecular wt, were formed in response to the added 35S MuLV RNA in both systems. All three polypeptides were precipitable with antiserum to detergent-disrupted MuLV. Finger-print analysis of tryptic digests indicated that all three contain amino acid sequences in common with each other and with the major methionine-containing structural proteins of the virion. This common peptide sequence suggests that these are overlapping polypeptides extending different distances from the same initiation site on the RNA. The small increase in the complexity of the map of 180K protein over those of the smaller ones is explained by aggregation, tandem repetition of the information for the 60K sequences in at least one of the virion RNAs, or the fact that the sequences additional to the 60K sequence in the 180K polypeptide are relatively poor in methionine. The last explanation is currently favored, particularly as the presence of additional non-methionine-containing MuLV-specific amino acid sequences in the 180K product cannot be ruled out at this stage. Further analyses and identification of all the sequences in this high-molecular wt polypeptide are needed before any of these explanations can be confirmed.

4459 DIFFERENTIAL CELLULAR REGULATION OF THREE DISTINCT CLASSES OF TYPE C RNA VIRUSES ENDOGENOUS TO MOUSE CELLS.

(Eng.) Aaronson, S. A.; Stephenson, J. R. *In: Tumor Viruses. Cold Spring Harbor Symposia on Quantitative Biology*. Cold Spring Harbor Lab. (Cold Spring Harbor, NY): vol. 39, pp. 1129-1137; 1975.

Several prototype mouse strains, including NIH Swiss, BALB/c, and C58, were studied to elucidate the role of endogenous viruses and genetic factors that affect virus expression in naturally occurring diseases. The endogenous viruses isolated from each prototype strain differ in terms of host range, neutralization by natural sera, and p12 antigenic determinant specificity. C58-MuLV and BALB:virus-1 are representative of class I viruses, BALB:virus-2 is representative of class II viruses, and NIH and NZB are characteristic of class III viruses. The two chemical inducers of type C viruses endogenous to mouse cells, halogenated pyrimidines and inhibitors of protein synthesis, show differences in the induction kinetics of the same virus, BALB:virus-2. The two inducers also differ in the efficiency with which they activate different classes of endogenous virus. The class II viruses were induced by both inducers with high efficiency, but the class I viruses were activated at high efficiency by iododeoxyuridine and at much lower frequency by cycloheximide. Class III viruses appeared completely resistant to induction by either class of chemicals. The *Fv-1* regulatory locus markedly influenced the establishment of the class I virus in transformed nonproducer cells following chemical activation. This regulatory locus had no apparent effect on class II endogenous viruses. The magnitude of virus release by individual embryo lines of (NIH x NZB)F_2 hybrids and backcross generations was investigated. The results agreed with those expected for the Mendelian segregation of a partially dominant locus influencing spontaneous expression of a xenotropic endogenous type C virus present in both NZB and NIH mouse cells. Indications are that the class I endogenous virus of C58 mouse cells and BALB:virus-1 are oncogenic. A genetic approach should aid in determining the biologic functions of class II and III endogenous viruses. (45 references)

4460 GENETIC CONTROL OF THE EXPRESSION OF MURINE LEUKEMIA VIRUS PROTEINS IN TISSUES OF NORMAL, YOUNG ADULT MICE. (Eng.) Strand, M.; Lilly, F.; August, J. T. *In: Tumor Viruses. Cold Spring Harbor Symposia on Quantitative Biology*. Cold Spring Harbor Lab. (Cold Spring Harbor, NY): vol. 39, pp. 1117-1122; 1975.

The occurrence of viral gene products in normal murine tissues was investigated. Radioimmunoassay techniques were used to measure the tissue concentrations of two of the major protein components of murine leukemia virus (MuLV) particles in selected inbred strains and crosses. The components are the internal protein p30 and the surface glycoprotein gp69/71. Mice of the high leukemia strains (AKR, C58/J, and PL/J) showed high concentrations of both p30 and gp69/71 in their tissues. Their concentration ratios in the tissues approximated those found in preparations of purified MuLV particles; i.e., twice as much p30 as gp69/71. Studies of F_1 crosses between AKR and C57L or BALB/c mice and of backcrosses suggested that the genetic determinants of the p30 molecule are not exclusively associated with the "V" genes *Akv-1* and *Akv-2* in the AKR genome. DBA/2 mice, which show only a low incidence of spontaneous leukemia, showed moderate amounts of p30 and high levels of gp69/71, whereas DBA/2-*Fv-1*b mice showed comparatively increased levels of p30 and decreased levels of gp69/71. The levels of p30 and gp69/71 corresponded to coordinate expression (2:1 ratio in respective activities) in the case of DBA/2-Fv-1b but not DBA/2. Low levels of both proteins were observed in four of five strains of the B10 congenic series; there was considerable deviation from the ratio expected with coordinate expression. BALB/c and congenic strains showed low levels of p30 and only trace amounts of gp69/71; nude homozygotes showed a dramatic increase in gp69/71 activity. Low to moderate levels of both proteins were detectable in 129, B6, and B6-G+IX mice, but 129-G-IX mice showed neither protein. The data indicate the existence of both structural and regulatory mouse genes that govern the expression of these substances. (18 references)

4461 HEREDITY OF THE GIX THYMOCYTE ANTIGEN ASSOCIATED WITH MURINE LEUKEMIA VIRUS: SEGREGATION DATA SIMULATING GENETIC LINKAGE. (Eng.) Stockert, E. (Memorial Sloan-Kettering Cancer Center, 1275 York Ave., New York, NY 10021); Boyse, E. A.; Sato, H.; Itakura, K. *Proc Natl Acad Sci USA* 73(6): 2077-2081; 1976.

The GIX antigen is a feature of the gp70 envelope glycoprotein of murine leukemia virus (MuLV). This GIX-gp70 molecule is found on the thymocytes of some (GIX+) strains of mice, where its expression is controlled by two mendelian genes, *GV-1* and *Gv-2*. Previous recombination data involving the prototype GIX+ mouse strain 129 indicated that the *H-2*(chromosome 17) and *Gv-1* loci are linked at a distance of 36 units from one another. In recombination studies reported here, the association of H-2 and GIX phenotypes is shown to be an example of quasilinkage, evidently dependent on heterozygosity at a locus or loci in the vicinity of *H-2*. Other previous recombination data, involving the GIX+ strain AKR, had indicated that the *Gpd-1* (chromosome 4) and *Gv-1* loci are linked at a distance of 19 units. Data are presented from other crosses that show that this association of Gpd-1 (glucose-6-phosphate dehydrogenase 1) and GIX phenotypes also constitutes quasilinkage, evidently due to heterozygosity at the *Fv-1* locus. An important theoretical consequence of quasilinkage in general is that it should enhance the heritability of particular constellations of unlinked genes, and so influence population structure. The data are discussed from the viewpoint that MuLV genomes are apparently involved in quasilinkage, and therefore may influence the genetic structure of populations. This in turn may strengthen the view that integrated MuLV genomes are not simply intruded into a self-sufficient cellular genome, but are themselves elements of the cellular genome with primary functions, perhaps in reproduction or embryogenesis. (24 references)

4462 ANTIGENIC ANALYSIS OF THE MAJOR STRUCTURAL PROTEIN OF THE MASON-PFIZER MONKEY VIRUS. (Eng.) Schochetman, G. (Meloy Labs., Inc., Springfield, VA 22151); Boehm-Truitt, M.; Schlom, J. *J Immunol* 117(1): 168-173; 1976.

Studies were performed to measure the antigenic cross-reactivity of a major structural protein of an RNA tumor virus with other similar viruses. The internal protein, p27 (molecular wt 27,000 daltons) of the Mason-Pfizer monkey virus (MPMV) was purified by gel filtration and ion-exchange chromatography and then used to develop a radioimmunoassay (RIA). This RIA was specific for MPMV because no immunologic cross-reactivity was observed between p27 of MPMV and 13 different RNA tumor viruses of mammalian and avian origin. However, the p27 of MPMV grown in three different primate cells exhibited identical antigenic cross-reactivity. In addition, significant levels of p27 were found only in MPMV-infected cells. These results indicate that synthesis of p27 is induced after virus infection and that p27 represents a viral-coded protein. (38 references)

4463 PRIMATE TYPE-C VIRUS NUCLEIC ACID SEQUENCES (WOOLLY MONKEY AND BABOON TYPES) IN TISSUES FROM A PATIENT WITH ACUTE MYELOGENOUS LEUKEMIA AND IN VIRUSES ISOLATED FROM CULTURED CELLS OF THE SAME PATIENT. (Eng.) Reitz, M. S. (Bionetics Res. Labs., Bethesda, MD 20014); Miller, N. R.; Wong-Staal, F.; Gallagher, R. E.; Gallo, R. C.; Gillespie, D. H. *Proc Natl Acad Sci USA* 73(6): 2113-2117; 1976.

Cultured peripheral blood leukocytes from a woman (patient HL23) with acute myelogenous leukemia produced type C RNA tumor viruses (HL23V). The viruses were analyzed by molecular hybridization after they were transmitted to five secondary cell culture lines. All of the transmitted virus isolates had nucleotide sequences related to the genome of simian sarcoma virus (SiSV). In addition, some transmitted viruses also had nucleotide sequences related to those of the baboon endogenous virus (BaEV). Molecular hybridization was also used to ascertain whether both viruses could have originated from patient HL23. With the use of ^{3}H-cDNA complementary to RNA from the separated BaEV-related component of HL23V and by hybridization of this cDNA to DNA from tissues of the patient, sequences related to BaEV were detected in the DNA obtained from the patient's spleen. However, no SiSV-related sequences were detectable. Cytoplasmic virallike particles, which had a buoyant density of 1.15-1.2 g/ml and were capable of synthesizing cDNA in association with a 35S RNA in vitro, were also found in the patient's fresh uncultured leukemic blood cells. cDNA synthesized by the cytoplasmic particles contained some sequences that hybridized to RNA from SiSv and, in addition, some that hybridized to RNA from BaEV. The cDNA also hybridized significantly to DNA isolated from the spleen of patient HL23 and to cytoplasmic RNA from the patient's leukocytes. The results suggest that the virus came directly from the leukemic cell samples. The finding of BaEV-related DNA proviral sequences in the spleen strongly supports this interpretation. The failure so far to find a complete SiSV-related provirus is perplexing, but could be attributable to the existence of such a provirus in DNA of only a small cell population in most leukemic patients. (33 references)

4464 CHARACTERIZATION OF SIMIAN CELLS TRANSFORMED BY TEMPERATURE-SENSITIVE MUTANTS OF SIMIAN VIRUS 40. (Eng.) Noonan, C. A. (Baylor Coll. Medicine, Houston, TX 77030); Brugge, J. S.; Butel, J. S. *J Virol* 18(3): 1106-1119; 1976.

An attempt was made to characterize simian cells that had been transformed by temperature sensitive mutants of wild-type simian virus 40 (SV40), Baylor reference strain. Seven lines derived from primary African green monkey kidney cells (GMK/WT, /A28, /B401, /B409, /B410, /B415, and /B416) were established. These cultures synthesized SV40 tumor (T) antigen constitutively, indicating a stable relationship between cellular and viral genomes. The cells released small amounts of virus even at high passage levels but eventually became negative for the spontaneous release of virus. Virus rescued from such "nonproducer" cells by transfection exhibited the growth properties of the original inoculum virus. Four of the cell lines were tested for the presence of altered growth patterns commonly associated with SV40-induced transformation. Although each of the cell lines was greater than 99% positive for T antigen, none of the cultures could be distinguished from primary or stable lines of normal simian cells on the basis of morphology, saturation density in high or low serum concentrations, colony formation on plastic or in soft agar, hexose transport, or concanavalin A agglutinability. However, the cells could be distinguished from the parental green monkey kidney cells by a prolonged life span, the presence of T antigen, a resistance to the replication of superinfection SV40 virus or SV40 viral DNA, and, with three of the four lines, an ability to complement the growth of human adenovirus type 7. These properties were expressed independent of the temperature of incubation. These results indicate that the presence of an immunologically reactive SV40 T antigen is not sufficient to ensure induction of phenotypic transformation and suggest that a specific interaction between viral and cellular genes and/or gene products may be a necessary requirement. A viral gene A product is required to maintain the transformed phenotype in a number of cell types, and it is possible that the T antigen in these cells is that product.

4465 ALTERED PATTERNS OF PROTEIN SYNTHESIS IN INFECTION BY SV40 MUTANTS. (Eng.) Tegtmeyer, P. *In: Tumor Viruses. Cold Spring Harbor Symposia on Quantitative Biology.* Cold Spring Harbor Lab. (Cold Spring Harbor, NY): vol. 39, pp. 9-15; 1975.

In vivo protein synthesis in infection by early and late temperature-sensitive mutants of SV40 was studied; infection by the early A mutants and the late mutant B11 was emphasized. Protein synthesis patterns in cells infected by wild-type and mutant virus were compared by short periods of 35S-methionine labeling. Protein synthesis patterns in uninfected cells varied in the relative quantities of individual proteins synthesized (depending upon incubation temperature), length of the radiolabeling period, and cell growth state. Except for inducing the synthesis of virion proteins (VP) and cellular histones, infection by wild-type virus resulted in only minor changes from the pattern in uninfected cells. Both the intracellular distribution and stability of VP1 are altered in infection by B11, suggesting that the major capsid protein may be the altered protein of the mutant. The most striking alteration in B11 infection was increased synthesis of specific nonvirion proteins, apparently of cellular origin. The most significant finding in A mutant infection at 41 C was the induction of a high molecular wt (100,000 daltons) protein with a rapid intracellular turnover that was identifiable after short periods of radiolabeling. Cellular extracts (known to contain induced proteins) were exposed to hamster anti-T serum to immunoprecipitate proteins possibly related to early viral function and to transformation. None of the B11-induced proteins was precipitated. The A58 100,000-dalton protein was detected in cells infected with wild-type virus or B11 but not in uninfected cells. It is suggested that this protein may be the only early viral protein; it may regulate its own synthesis since it is overproduced in A mutant infection. (21 references)

4466 DEATH AND TRANSFORMATION. (Eng.) Martin, R. G.; Anderson, J. L. *In: Biology of Radiation Carcinogenesis*. Edited by Y. M. Yuhas; R. W. Tennant; J. D. Regan. (New York: Raven Press): pp. 287-300; 1976.

The role of SV40 in inducing transformation in tissue cultures of the Chinese hamster lung (CHL) was examined. CHL cells were infected with *A*, *B*, *C*, *BC*, or *D* mutants of SV40 and grown on 2.5% serum. All transformants behaved like control wild-type SV40 transformed cells at the permissive temperature (33 C) and differently from normal, uninfected cells, but the *A* mutants (*A207* and *A239*) acted like normal cells at the nonpermissive temperature (40 C), indicating that the *A* function is required for rapid growth in low serum concentrations. T antigen (assayed by indirect immunofluorescence) is not contained in normal cells, but all mutants (*A207*, *A209*, *A239*, *A241*, *B201*, *BC245*, *C219*, and *D202*) exhibited T antigen at permissive and nonpermissive temperatures. The uptake of 2-deoxy glucose is not related to the other manifestations of SV40-transformed CHL and mouse brain cells. In relatively poor medium (Ham's F-12) at 33 C, wild-type and *A239*-transformed cells grow more rapidly than normal CHL cells and they grow beyond a cell density of 10^5 cells/cm^2. Neither fully transformed growth at 33 C nor temperature-sensitive ''revertant'' behavior at 40 C depends on accompanying changes in chromosome number. A mechanism for SV40 transformation was proposed in which the degree of transformation can be explained by position effects on the expression of the SV40 genome; e.g., cells will be minimally transformed when SV40 is integrated in chromosome regions normally ''turned off'' and cells will be fully transformed when SV40 is integrated in chromosome regions expressed constitutively.

4467 AMINO ACID AND HEXOSE TRANSPORT OF NORMAL AND SIMIAN VIRUS 40-TRANSFORMED HUMAN CELLS. (Eng.) Patterson, M. K., Jr. (Biomedical Div., Samuel Roberts Noble Foundation, Inc., Ardmore, OK 73401); Birckbichler, P. J.; Conway, E.; Orr, G. R. *Cancer Res* 36(2): 394-397; 1976.

Studies on amino acid and hexose transport were performed on human WI-38 cells and WI-38 SV40-transformed cells (VA13A and VA13-2RA). Depending upon cell line or conditions, either no difference or a relative decrease in initial uptake by transformed cells was found. Under similar growth conditions, transformed hamster cells (PyBHK-21/C13) had increased uptake, compared with the normal hamster cells (BHK-21/C13). The normal and transformed human cells were also similar in sialic acid content and agglutinated when treated with concanavalin A.

4468 IN VITRO TRANSFORMATION OF HUMAN PROSTATIC CARCINOMA. (Eng.) Jones, R. E. (Dept. Surgery and Microbiology, M. S. Hershey Medical Center, Pennsylvania State Univ. Coll. Medicine, Hershey, PA); Sanford, E. J.; Rohner, T. J., Jr.; Rapp, F. *J Urol* 115(1): 82-85; 1976.

Cell cultures derived from fetal human and prostatic adenocarcinoma were inoculated with SV40 virus. Within six wk in vitro morphologic transformation occurred, after which the cells proliferated rapidly. They exhibited the SV40 T-antigen by immunofluorescence and survived propagation beyond the limited lifespan of uninfected cultures. It is suggested that this may be a useful technique in establishing stable cell lines of human prostate.

4469 REGULATION OF VIRAL TRANSCRIPTION AND TUMOR ANTIGEN EXPRESSION IN CELLS TRANSFORMED BY SIMIAN VIRUS 40. (Eng.) Basilico, C. (Dept. Pathology, New York Univ. Sch. Medicine, New York, NY 10016); Zouzias, D. *Proc Natl Acad Sci USA* 73(6): 1931-1935; 1976.

The expression of simian virus 40 (SV40) specific tumor antigen (T antigen) and viral RNA in SV40-transformed mouse 3T3 cells that are temperature-sensitive for expression of the transformed phenotype (ts SV3T3) was studied. Although transformed by wild-type SV40, ts SV3T3 cells at 32 C behaved like standard transformants, but at 39 C they became arrested in G_1 after reaching saturation density or under conditions of serum starvation. ts SV3T3 cells at 32 C or exponentially growing at 39 C were uniformly T antigen positive. However, after G_1 arrest at 39 C the majority of the cells became T antigen negative. Induction of proliferation in the resting cultures resulted in the reappearance of T antigen in most of the cells, concomitant with the induction of DNA synthesis. The reason for the disappearance of T antigen from ts SV3T3 cells arrested in G_1 seemed to reside in a transcriptional control operating on the integrated viral DNA, since these cells contained no appreciable amounts of SV40 specific RNA. Viral RNA can be easily detected in cells growing at 32 or 39 C. The results suggest that viral genome transcription in SV40-transformed cells is cell cycle-dependent. (30 references)

4470 STEPWISE RELAXATION OF SUPERCOILED SV40 DNA. (Eng.) Keller, W.; Wendel, I. *In: Tumor Viruses. Cold Spring Harbor Symposia on Quantitative Biology*. Cold Spring Harbor Lab. (Cold Spring Harbor, NY): vol. 39, pp. 199-208; 1975.

Agarose gel electrophoresis was used to examine the tertiary structure of supercoiled simian virus 40 (SV40) DNA. In addition, the number of superhelical turns in SV40 DNA was determined by counting the number of intermediates found at early times in the reaction of DNA with the relapsing activity. Differences as small as one superhelical turn were detected by this method. When it was used as an assay, a protein could be purified from human tissue culture cells that acts on supercoiled DNA, leading to a gradual removal of its superhelical turns. When the purified DNA relaxing protein was used to generate reaction intermediates of DNAs with different numbers of superhelical turns, it was found that native SV40 DNA contains a minimum of 20 physical superhelical turns. The upper limit of this number is less accurately defined, but it is probably not higher than 24. These numbers agree with the numbers determined previously by various indirect physical methods. (21 references)

4471 MAPPING OF SV40 AND mRNA SPECIES ON THE VIRAL GENOME. (Eng.) Weinberg, R. A.; Newbold, J. E. *In: Tumor Viruses. Cold Spring Harbor Symposia on Quantitative Biology.* Cold Spring Harbor Lab. (Cold Spring Harbor, NY): vol. 39, pp. 161-164; 1975.

Studies were undertaken to map the early RNA, large late RNA, and the small late RNA of SV40. Aliquots of RNA were hybridized to cleavage fragments of the DNA genome immobilized on cellulose nitrate filters. The fragments were derived by cleaving SV40 Form I DNA with restriction endonuclease Z isolated from *Haemophilus aegyptius*. Evidence is presented that allows the construction of a tentative map of the sites on the SV40 genome from which the various transcripts are derived. Technical difficulties in hybridizing RNA to the small genome fragments prevented the exact localization of the 5' ends of the three RNA species. Also, the assignment of the 3' ends of the two late RNAs as exactly coterminous remains to be shown.

4472 PROPERTIES OF REPLICATING SV40 DNA MOLECULES AND MAPPING UNPAIRED REGIONS IN SV40 DNA I. (Eng.) Salzman, N. P.; Lebowitz, J.; Chen, M.; Sebring, E.; Garon, C. F. *In: Tumor Viruses. Cold Spring Harbor Symposia on Quantitative Biology.* Cold Spring Harbor Lab. (Cold Spring Harbor, NY): vol. 39, pp. 209-218; 1975.

Analysis of replicating DNA molecules of simian virus 40 (SV40) fractionated according to the extent of replication is reported. After newly synthesized strands were dissociated from replicating molecules, covalently closed duplexes were generated. This provides direct evidence for the processes of nicking, unwinding, and sealing of parental strands during DNA replication. Analyses of replicating DNA molecules at different stages of replication by sedimentation velocity-dye titration demonstrated that the superhelix density of the unreplicated portion of the replicating molecules was greater than that of DNA I (SV40 component I, the superhelical form) and that it increased as the degree of replication increased. The increase in superhelix density over that of DNA I corresponded to a rather constant two to five additional superhelical turns per molecule, regardless of the extent of replication. This suggests that a region (or regions) of about 20 to 50 nucleotides may exist in a denatured state in replicating molecules, presumably at the replicating forks of the molecule. The presence of single-stranded regions in SV40 DNA I was determined to see whether they play some role in the binding of proteins to DNA. Three single-stranded regions were localized in *Hemophilus influenzae* restriction endonuclease fragments A, B, and G, using N-cyclohexyl-N'-β-(4-methylmorpholinium)-ethyl carbodiimide p-toluene sulfonate binding to SV40 DNA I. (40 references)

4473 FUCOLIPID PATTERNS OF CELL LINES TRANSFORMED BY HIGHLY AND WEAKLY TUMORIGENIC SIMIAN VIRUS 40 AND HERPES SIMPLEX VIRUS. (Eng.) Steiner, S. (Baylor Coll. Medicine, Houston, TX 77030); Steiner, M. R. *Intervirology* 6(1): 32-41; 1975/76.

The fucolipids of simian virus 40 (SV40)-transformed Balb/c mouse cells (MKS, Tu-MKS, 3T3, SV3T3, K-Balb, and TuT-3) and herpes simplex virus (HSV)-transformed hamster cells (333-8-9 and MS 4-1) were examined. Cultures were seeded at low density in medium supplemented with either ^{3}H-fucose or ^{14}C-fucose and incubated until confluent (76-96 hr). In the virus-transformed cell lines and in a spontaneously transformed cell line there was a decrease in the incorporation of fucose radioactivity into fucolipid IV, the least chromatographically mobile, most complex fucolipid. Only in the highly transplantable SV40- and HSV-transformed cell lines was the decrease in incorporation of radioactivity in fucolipid IV as marked as that observed in oncornavirus-transformed cell lines. Similarly, only in the highly tumorigenic DNA-virus-transformed cell lines K-Balb, TuT-3, and Tu-MKS) was there a significant increase in incorporation of radioactivity from radioisotopically labeled fucose into fucolipid III, as has been observed in oncornavirus-transformed cell lines. The results suggest that fucolipids may be a useful marker of cellular transformation.

4474 STUDIES ON IN VITRO TRANSFORMATION BY DNA AND DNA FRAGMENTS OF HUMAN ADENOVIRUSES AND SIMIAN VIRUS 40. (Eng.) Graham, F. L.; Abrahams, P. J.; Mulder, C.; Heijneker, H. L.; Warnaar, S. O.; de Vries, F. A. J.; Fiers, W.; van der Eb, A. J. *In: Tumor Viruses. Cold Spring Harbor Symposia on Quantitative Biology.* Cold Spring Harbor Lab. (Cold Spring Harbor, NY): vol. 39, pp. 637-650; 1975.

Attempts were made to locate and isolate segments of adenovirus and SV40 DNA that are able to transform cells in vitro. DNA fragments were prepared by mechanical shear or by incubation with restriction endonucleases; single-stranded ends were generated by exonuclease III digestion. Transformation assays were made with primary rat kidney cell cultures using the calcium assay technique. Noninfectious, mechanically broken DNA fragments retained their transforming capacity, even fragments as small as 3 x 10^6 daltons or one-eighth of the adenovirus 5 genome. DNA fragments sedimenting slower than approximately 10S had no transforming activity. The length of the transforming segment was not greater than about 1 x 10^6 daltons, or 4%-5% of the adenovirus 5 genome. The transforming segment begins at 1.1% from the left terminus of the molecule and terminates at about 5%-6%, about 350 base pairs from the terminus. A specific fragment with transforming activity could be isolated from adenovirus 2 and 5 DNA digested with endonuclease R·*HsuI*. This fragment had a molecular wt of 1.6 x 10^6 daltons and represented the left terminal fragment of both DNAs. Primary hamster kidney cells (which are permissive for adenovirus 5) were readily transformed by shear-induced fragments of adenovirus 5 DNA; human embryonic kidney cells were much less readily transformed. Hamster cells transformed by adenovirus 5 DNA produced tumors in newborn hamsters, while similarly transformed rat kidney cells produced tumors in newborn isologous rats. The transformed cell lines were positive for adenovirus T antigen. With the calcium technique, SV40 DNA transformed both primary rat kidney cells and mouse 3T3 cells. The rat cells were also transformed with linear molecules of SV40 DNA and with SV40 DNA fragments. (40 references)

4475 CONSTRUCTION OF THE GENETIC MAP OF THE POLYOMA GENOME. (Eng.) Miller, L. K. (Imperial Cancer Res. Fund, Lincoln's Inn Fields, London WC2A 3PX, England); Fried, M. *J Virol* 18(3): 824-832; 1976.

Because of the low recombination frequencies and lack of distinct lethal mutations for the polyoma genome, attempts were made to map seven early mutants, three late mutants, and one plaque morphology mutant by use of restriction endonuclease fragments in a maker rescue technique. The early mutants mapped between 1.0 and 26.4 U from the *Eco*RI site, a region previously shown to correspond to the 3'-OH terminal half of ''early'' RNA. The late mutants as well as the plaque morphology mutant mapped between 26.6 and 45.4 U, a region previously shown to correspond to the 3'-OH terminal half of ''late'' RNA. Analysis of the genotype of rescued virus demonstrated that the modification of the mutant DNA during marker rescue was limited to the region of the genome covered by the wild-type restriction endonuclease fragment tested.

4476 AIRBORNE TRANSMISSION OF POLYOMA VIRUS. (Eng.) McGarrity, G. J. (Dept. Microbiology, Inst. Medical Res., Camden, NJ 08103); Coriell, L. L.; Ammen*, V. *J Natl Cancer Inst* 56(1): 159-162; 1976.

Polyoma virus (PV) infection was transmitted through the air of an animal laboratory. Mice free of detectable antibodies to PV were exposed for one month to the airborne environment of laboratories housing naturally infected mice. The seroconversion rate was 75% (24/32), as measured by hemagglutination inhibition. Control mice, housed in the sterile atmosphere of a mass air flow cabinet (MAFC) in the same laboratory, had a seroconversion rate of 15.8% (3/19). Airborne transmission occurred via PV aerosols, generated by the handling of contaminated bedding, cages and mice during weekly housekeeping. Length of exposure to PV aerosols correlated with seroconversion. One- and three-hour exposures resulted in seroconversion rates of 40% (6/15) and 72% (23/32), respectively. Seroconversion rates of mice continuously housed in MAFC totaled 5% (2/40). Checkerboarding mice free of detectable antibodies with mice given 10^5 mean tissue culture infective doses of PV ip resulted in an airborne infection rate of 50% (15/30) in a conventionally ventilated room during a 12 wk study. The airborne transmission rate was 10% (3/30) when experiments were performed in a mass air flow room with a vertical air velocity of 26 feet/min, and 0% (0/30) when a vertical air velocity of 30 feet/min was used. Antibodies to PV could not be detected in any of 138 human sera, including sera from 29 animal-care technicians who handled PV-infected mice and 15 personnel who had worked with the virus.

4477 A SIMPLIFYING CONCEPT IN TUMOR VIROLOGY: VIRUS-SPECIFIC 'PLEIOTROPIC EFFECTORS.' (Eng.) Weil, R.; Salomon, C.; May, E.; May, P. *In: Tumor Viruses. Cold Spring Harbor Symposia on Quantitative Biology.* Cold Spring Harbor Lab. (Cold Spring Harbor, NY): vol. 39, pp. 381-395; 1975.

Transcription of simian virus 40 (SV40) and polyoma DNA during lytic infection and abortive infection and in stably transformed cells are discussed.
A model that reconciles the paradox of the many biological effects of polyoma and SV40 with their small amount of genetic information and satisfactorily accounts for experimental observations is presented. In confluent primary mouse kidney cell cultures, abortive and lytic infection with SV40 and polyoma, respectively, showed a significant degree of analogy. The observations suggest that synthesis of virus-specific 19S mRNA and of T antigen does not require integration of parental viral DNA and that it is carried out by host cell enzymes present before infection. The observations are compatible with the hypothesis that infection remains abortive if the virus, though able to stimulate chromatin replication, cannot induce the additional functions required for progeny virus production. Polyoma- and Av40-specific early 19S mRNAs are probably the contiguous transcript of the early region of the viral genome, comprising 40%-45% of the early strand. Acquisition by cells of a transformed phenotype is not synonymous with conversion to cancer cells; it is suggested that all animal cells carry genes that specify pleiotropic effectors (polypeptides that play an important role in regulation of phenotypic expression and of mitotic activity). (74 references)

4478 THE FLUIDITY OF NORMAL AND VIRUS-TRANSFORMED CELL PLASMA MEMBRANE. (Eng.) Micklem, K. J. (Dept. Biochemistry, Univ. Oxford, South Parks Road, Oxford OX1 3QU, UK); Abra, R. M.; Knutton, S.; Graham, J. M.; Pasternak, C. A. *Biochem J* 154(3): 561-566; 1976.

The phospholipid composition of the plasma membrane of two closely related cells, normal and polyoma virus-transformed baby hamster kidney (BHK) cells, was compared in detail, the relative turnover of acyl-labeled phospholipids was assessed, and the distribution of intramembranous particles was examined. The cells were disrupted by nitrogen cavitation and subcellular organelles were isolated. Phospholipids were separated by two-dimensional chromatography on silica gel and detected by radioautography or by exposure to iodine vapor. Selected areas were extracted and analyzed by gas-liquid chromatography or, for labeled phosphatidylcholine, by argentation chromatography. For electron microscopy, cell cultures were fixed with glutaraldehyde, in some cases after being incubated for 10 min with concanavalin A (100 μg/ml, Con A). The phospholipid composition and cholesterol/phospholipid ratio of plasma membrane were the same in normal as in transformed BHK cells; no significant difference in length or degree of unsaturation of the contributing acyl chains was apparent. In each case, phosphatidylcholine and phosphatidylethanolamine accounted for more than 60% of all phospholipids. The turnover of acetate-labeled phosphatidylcholine species in the plasma membrane of normal and transformed BHK cells was also the same. When normal and transformed 3T3 cells, in sparse and confluent cultures, were examined after fixation at 4 and 37 C by freeze fracture electron microscopy, a random distribution of intramembranous particles was seen. There was no correlation between distribution of particles and movement of Con A receptor sites. It is concluded that increased mobility of lectin-receptor sites on transformed fibroblasts cannot be ascribed to an alteration in the composition or turnover of the plasma-membrane lipids. (57 references)

4479 GENETIC ANALYSIS OF HUMAN ADENOVIRUS TYPE 5 IN PERMISSIVE AND NONPERMISSIVE CELLS. (Eng.) Williams, J. F.; Young, C. S. H.; Austin, P. E. *In: Tumor Viruses. Cold Spring Harbor Symposia on Quantitative Biology*. Cold Spring Harbor Lab. (Cold Spring Harbor, NY): vol. 39, pp. 427-437; 1975.

Recent results concerning the genetic analysis of human adenovirus type 5 (Ad5) using temperature-sensitive (ts) mutants, and results relating the interaction of ts mutants with other viruses and to the ability of mutants to transform rat embryo cells are presented. An upper limit of about 10^{-4} was estimated for most of the ts mutants. On the basis of complementation tests, 51 mutants were classified into five functional groups. Complementary crosses between Ad5 and Ad12 produced viruses possessing antigenic determinants from both parental types; this double antigenicity was due to phenotypic mixing, not genetic recombination. Most Ad5 mutants were not enhanced at all by simian virus 40 (SV40) in complementation tests, but mutants of five complementation groups showed varying degrees of enhancement. A tentative ts mutant genetic map was constructed, and the following conclusions were drawn from the data obtained: stable recombinants are formed in crosses between different mutants; recombination frequencies between different ts mutants are not random, and markers can be ordered in a basic additive sequence. Noncomplementary mutants show very low recombination frequencies; mutants showing low complementation indices also show low recombination frequencies; and, mutants that complement well show higher recombination frequencies. The ability of Ad5 (wild-type and ts mutants) to transform rat embryo cells was investigated. Recent evidence indicates that the genes necessary for maintaining the transformed state of rat cells must reside in a 14% segment located at the left end of the genome. (44 references)

4480 PROPERTIES AND BEHAVIOR OF HAMSTER EMBRYO CELLS TRANSFORMED BY HUMAN ADENOVIRUS TYPE 5. (Eng.) Goldman, R. D.; Chang, C.; Williams, J. F. *In: Tumor Viruses. Cold Spring Harbor Symposia on Quantitative Biology*. Cold Spring Harbor Lab. (Cold Spring Harbor, NY): vol. 39, pp. 601-614; 1975.

Differences in the motile behavior of normal hamster embryo cells and their human adenovirus type 5-transformed derivatives were correlated to intracellular alterations in cytoplasmic fibers and to changes in cell surface architecture. Light, transmission electron, and scanning electron microscopy were employed. The normal cells grew in flattened configurations and generally possessed an epithelioid shape. They showed membrane ruffling and macropinocytosis and were capable of locomotion across a substrate. Prior to division, the cells became rounded and assumed a spherical shape; blebbing activity appeared and then disappeared within 15-60 min. The daughter cells were capable of locomotion. The transformed cells failed to spread on the substrate, retained a rounded morphology, possessed highly motile blebs at their surface, showed neither ruffling nor macropinocytosis, and were incapable of locomotion. The blebbing activity remained after cytokinesis; the daughter cells blebbed and remained rounded and stationary during interphase. The transformed cells lacked microfilament bundles throughout the cytoplasm and contained only a few randomly distributed microtubules and 100-A filaments. The cell surfaces contained few microvilli. These cells tended to pile up on each other rather than fill in the adjacent spaces. The addition of dibutyryl cAMP (0.5-1.0 M) in the presence of 1.0 mM theophylline caused the transformants to flatten significantly, lose their blebbing activity, form long cell processes, and assume locomotor capabilities. Microtubules, microfilament bundles, and 100-A filaments became more numerous. The cells reverted to their transformed morphology in a normal medium. The data indicate that human adenovirus 5 can produce transformants that phenotypically resemble normal dividing cells in many ways. (29 references)

4481 ADENOVIRUS TYPE 12 INFECTION OF DEFINED MOUSE-HUMAN HYBRID CELL CLONES. (Eng.) Biron, K. K. (Rutgers Medical Sch., Piscataway, N.J. 08854); Raska jun, K. *Experientia* 32(1): 38-40; 1976.

The infection of specific clones of mouse-human cell hybrids was studied to determine whether the presence of certain human chromosomes in hybrid cells would make them permissive for adenovirus replication. Mouse cells, human cells, and mouse-human cell hybrids (Cl-1D mouse cells hybridized to either KOP-2 human fibroblasts or to a line of simian virus 40-transformed cells derived from patients with the Lesch-Nyhan syndrome) were infected with adenovirus type 12 (Ad 12) at an input multiplicity of infection of 100 PFU/cell. Adenovirus-specific T-antigen was detected in 60% of mouse cells, in 90%-95% of human cells, and in 61%-92% of different clones of the hybrids. No virus capsid proteins were detected in mouse cells; positive staining was detected in 95% of human cells. The presence of virus-specific capsid proteins and newly synthesized virus-specific DNA was observed in two clones of hybrid cells. Both clones contained the human chromosomes A3, B5, C7, C11, C12, D14, E17, F19, and F20.

4482 ADENOVIRUS-INFECTED, CELL-SPECIFIC, DNA-BINDING PROTEINS. (Eng.) Levine, A. J.; van der Vliet, P. C.; Rosenwirth, B.; Rabek, J.; Frenkel, G.; Ensinger, M. *In: Tumor Viruses. Cold Spring Harbor Symposia on Quantitative Biology*. Cold Spring Harbor Lab. (Cold Spring Harbor, NY): vol. 39, pp. 559-566; 1975.

Cell-specific DNA-binding proteins were isolated from adenovirus-infected cells and characterized. African green monkey kidney (AGMK) cells were mock-infected or infected with type 5 adenovirus (Ad5) and labeled with ^{14}C-leucine or ^{3}H-leucine. Two infected, cell-specific, single-stranded DNA-binding proteins were produced with molecular wt of 72,000 and 48,000 daltons. Similar proteins were found in Ad2- and Ad12-infected AGMK cells. The purified DNA-binding proteins bound preferentially and cooperatively to any single-stranded DNA tested, but no binding to double-stranded DNA or 23S *Escherichia coli* RNA was detected. Approximately 1 to 10 x 10^{6} molecules were synthesized after infection of the AGMK cells, suggesting a stoichiometric rather than a catalytic function for these proteins. Extracts of Ad5 ts 125-infected AGMK cells prepared at the nonpermissive temperature did not contain detectable adenovirus DNA-binding proteins, but these proteins were present in extracts prepared at the permissive temperature. The amount of DNA-binding proteins consistently decreased when Ad5 ts 125-infected AGMK cells were shifted from the permissive to the nonpermissive temperature. The proteins produced at permissive temperatures appeared to be more thermolabile for binding to single-stranded DNA than wild-type proteins. It is possible that the 48,000-dalton protein is a proteolytic breakdown product of the 72,000-dalton protein. SV40 infection of AGMK cells was able to overcome the Ad5 ts 125 block to adenovirus DNA synthesis. SV40 ts B-8 and SV40 ts B-11 mutants, but not SV40 ts A-30, were also able to complement Ad5 ts 125 in AGMK cells. The data suggest that the adenovirus-induced DNA-binding proteins are required for adenovirus DNA replication. Common functional aspects of adenovirus and SV40 cellular transformation are also suggested. (24 references)

4483 ANTIBODIES TO EPSTEIN-BARR VIRUS CAPSID ANTIGENS IN MALIGNANT TUMORS OF CHILDREN. (Ger). Lubec, G. (Universitaets-Kinderklinik Wien, Waehringer Guertel 74-76, A-1090 Vienna, Austria); Krepler, P. *Paediatr Paedol* 11(1): 88-93; 1976.

The levels of antibodies to viral capsid antigens of Epstein-Barr virus in the sera of 25 healthy children and 25 children with leukemia or malignant tumors were determined by immunofluorescence microscopy. Leukemic patients showed mean arithmetic titers of 29.285, healthy children 24.8. Patients with malignant tumors showed higher titers, mean value 44.285. Because of the insignificant differences between titers in leukemic and healthy children, no correlation of tumor mass or therapy and antigen levels could be established.

4484 BIOLOGICAL PROPERTIES AND VIRAL SURFACE ANTIGENS OF BURKITT LYMPHOMA- AND MONONUCLEOSIS-DERIVED STRAINS OF EPSTEIN-BARR VIRUS RELEASED FROM TRANSFORMED MARMOSET CELLS. (Eng.) Miller, G. (Sch. Medicine, Yale Univ., New Haven, CT 06510); Coope, D.; Niederman, J.; Pagano, J. *J Virol* 18(3): 1071-1080; 1976.

An attempt was made to analyze the homogeneity and heterogeneity of the biological properties of virus surface antigens of three strains of Epstein Barr virus (EBV): the Olare and Nyevu strains from Burkitt's lymphoma (BL) and the Hawley strain from infectious mononucleosis (IM). The three viruses shared properties of the transforming biotype of EBV; namely, stimulation of DNA synthesis and immortalization of cord blood leukocytes and failure to induce ''early antigen'' in lymphoblast lines. All viruses produced more virus in transformed marmoset cells than in transformed human cells. Reference human sera and sera from primary EBV infections were used to compare the three virus strains in a virus neutralization test based on inhibition of stimulation of DNA synthesis. Specimens taken late in convalescence from patients with mononucleosis and sera from marmosets experimentally infected with virus from a patient with mononucleosis neutralized the homologous virus, as well as the two virus strains isolated from patients with BL. This finding indicates that viral antigens that elicit neutralizing antibodies are shared among the strains. However, in certain sera the neutralizing-antibody titer against one strain was consistently higher than against another strain. Furthermore, sera taken early after onset of IM contained low levels of neutralizing antibody against IM-derived virus, but failed to neutralize NL-derived virus. These latter findings suggest the existence of heterogeneity among surface antigens of EBVs. The results emphasize the biological and antigenic similarity of EBV isolates from BL and IM. It is not known whether the described diversity or the genome variation is epidemiologically significant.

4485 TRANSFORMATION OF TONSIL LYMPHOCYTES BY EPSTEIN-BARR VIRUS. (Eng.) Yamamoto, N. (Dept. Microbiology, Kumamoto Univ. Medical Sch., Kumamoto, 860, Japan); Katsuki, T.; Hinuma, Y. *J Natl Cancer Inst* 56(6): 1105-1107; 1976.

Tonsil lymphocytes obtained from children and adults at tonsillectomy were examined for susceptibility to infection with Epstein-Barr virus (EBV) in vitro. Of 37 specimens, 24 (65%) were transformed by EBV. The transformation rate was higher in the cells from younger children (92% among children 5 to 10 yr old) than in those from older children and adults (41% to 62%), but the susceptibility was not directly correlated to the titer of antibody against EBV in donor serum. All 22 transformed tonsil cell lines tested were carrying EBV-associated nuclear antigen, and 8 of them produced cytoplasmic IgA in 15%-45% of the cells. The results suggest that tonsil lymphocytes are targets of EBV and may be important in the natural transmission of the virus. (27 references)

4486 EBV DNA IN NONLYMPHOID CELLS OF NASOPHARYNGEAL CARCINOMAS AND IN A MALIGNANT LYMPHOMA OBTAINED AFTER INOCULATION OF EBV INTO COTTONTOP MARMOSETS. (Eng.) Wolf, H.; Werner, J.; zur Hausen, H. *In: Tumor Viruses. Cold Spring Harbor Symposia on Quantitative Biology.* Cold Spring Harbor Lab. (Cold Spring Harbor, NY): vol. 39, pp. 791-796; 1975.

The occurrence of Epstein-Barr (EB) viral DNA in the nonlymphoid cells of nasopharyngeal carcinomas and in the tumor material from cottontop marmosets inoculated with EBV was studied. DNA from the biopsies of 34 nasopharyngeal carcinomas was hybridized with EBV cRNA (complementary RNA). In the primate experiments, marmosets were inoculated with neutralized or nonneutralized EBV concentrated from the supernatants of Kaplan (infectious mononucleosis) or P3HR-1 (Burkitt's tumor) cells. The animals were examined for the development of EBV antibodies by indirect immunofluorescence. All but three of the tumor materials were negative; upon histological reexamination, two were found not to contain tumor tissue and the third was shown to be an adenocarcinoma. The 11 highly hybridizing tumors were of predominantly epithelial morphology, whereas lymphatic cells represented a majority in most of the remaining biopsies. In situ hybridizations of cytological preparations further confirmed the specific association of EBV with epithelial nasopharyngeal carcinoma cells. In the primate experiments, one animal that had received the nonneutralized Kaplan virus developed a lymphoproliferative disease 7 wk after inoculation. DNA derived from a submandibular tumor contained slightly more than one genome equivalent of EBV DNA, and about 0.5 genome equivalent was found in the spleen DNA. None of the other marmosets developed tumors, and no rise in antibodies against EBV antigens was observed in any but the tumor-bearing animals.. (30 references)

4487 IMMORTALIZING AND NONIMMORTALIZING LABORATORY STRAINS OF EPSTEIN-BARR VIRUS. (Eng.) Miller, G.; Robinson, J.; Heston, L. *In: Tumor Viruses. Cold Spring Harbor Symposia on Quantitative Biology.* Cold Spring Harbor Lab. (Cold Spring Harbor, NY): vol. 39, pp. 773-781; 1975.

The expression of two biologic properties (superinfection and transformation or immortalization) by two laboratory strains of Epstein-Barr virus (EBV) was studied. Viruses were derived from the extracellular fluids of two continuous lymphoblastoid cell lines (P_3J-HR-1 and B95-8). Viral antigens were determined by indirect immunofluorescence, complement fixation, and gel precipitation, and an end point dilution assay was used to study immortalization. Assays by electron microscopy, superinfection of nonproducer cell lines, and stimulation of ^{3}H-thymidine incorporation into umbilical cord leukocytes were also used. The results with four different antigen systems (viral capsid antigen, EB nuclear antigen, a soluble antigen detected by complement fixation, and an antigen detected by immunodiffusion) illustrated the close antigenic similarity of the two EBV strains. However, although extracellular fluids from the B95 line contained approximately 10^5 50% immortalizing units/ml, a virus with immortalizing properties has never been found in the supernatant fluid of the P-H line. Similarly, although B95 virus stimulated the ^{3}H-thymidine incorporation into leukocytes, P-H virus did not. The P-H virus, but not the B95 virus, caused the appearance of early antigen in six lymphoblastoid cell lines that contained the EB viral genome. P-H virus interfered with B95 immortalization and stimulation of DNA synthesis; the interference appeared to be due to the virus itself rather than some soluble component in the extracellular fluid (such as interferon). These findings represent the first indication of heterogeneity in the EB virus group. (34 references)

4488 HERPESVIRUS ANTIGENS AND CELL-MEDIATED IMMUNITY IN CERVICAL CANCER. (Eng.) Aurelian, L.; Strand, B. C.; Jacobs, R. P.; Bell, R. B.; Smith, M. F. *In: Tumor Virus Infections and Immunity.* Edited by R. L. Crowell; H. Friedman; J. E. Prier. (Baltimore: Univ. Park Press): pp. 89- 131; 1976.

Patients with cervical cancer have antibody to herpes simplex virus type 2 (HSV-2) at a much higher frequency than matched controls, and cervical tumor cells contain HSV-2 DNA, viral RNA, and antigens, both structural and nonstructural. Some HSV-2-transformed cells can cause tumors in animals. Biopsies of cervical tumors indicate the production of an antigen (AG-4) that is also HSV-induced, and this antigen has been shown to be antigenically distinct from virion antigens and to be located on the cell surface. Autoradiography of virion proteins has indicated the presence of 24 virus-specific proteins. Infected HEp-2 cells synthesize virus-specific proteins. Complement-fixing ability with AG-4-positive cervical cancer sera may be associated with a virion polypeptide designated VP 4. Studies on cell-mediated immunity to herpesvirus are reviewed in terms of immunity in primary herpesvirus infections and that in latency. The possible involvement of the cellular response to viral antigens in cervical cancer might be explained by any of three hypotheses: an alteration of the immunologic surveillance system; the appearance on tumor cells of a tumor-specific, virus-coded antigen, which stimulates clones of antigen-reactive lymphocytes; tumor induction accompanied by the development of specific antibody populations that can act as a "blocking" antibody, either by binding to antigen at recognition sites or to the tumor-specific, virus-coded antigen on the tumor cells, rendering them inaccessible to effector lymphocytes. In patients with invasive cervical cancer, VP 4 is associated with impaired blastogenic responses; women with precancerous lesions do not exhibit a similar impairment. (54 references)

4489 ISOLATION OF EPSTEIN-BARR TYPE VIRUS FROM A LYMPHOBLASTOID CELL SUSPENSION CULTURE FROM A PATIENT WITH LYMPHOGRANULOMATOSIS. (Rus.) Lapin, B. A. (Inst. Experimental Pathology and Therapy, Acad. Medical Sciences, USSR, Sukhumi, USSR); Agrba, V. Z.; D'iachenko, A. G.; Iakovleva, L. A.; Sanguliia, I. A.; Kokosha, L. V.; Chuvirov, G. N.; Kakubava, V. V. *Dokl Akad Nauk SSR* 227(6): 1496-1499; 1976.

Tumor fragments from a 69-yr-old patient with mixed-cell lymphogranulomatosis were propagated in a suspension culture (ChSL-1). Optimal results were obtained when ChSL-1 cells were cultured in an atmosphere of 5% CO_2 or with constant agitation at 37 C. The morphology was relatively homogeneous, characterized primarily by lymphoblastoid elements with oval nuclei containing one to two nucleoli and fairly extensive, sometimes vacuolated, cytoplasm. Experiments with Syrian hamsters and white mice showed that the investigated virus did not possess tumorigenic or contagious properties and was not a herpes simplex virus. Immunofluorescence revealed 1.8%-3.2% of cells with clear granular cytoplasmic fluorescence, a reaction similar to that seen in the cytoplasm of cultured cells transformed by Epstein-Barr virus (EBV). Radioimmunoprecipitation using a ^{3}H-thymidine-labeled virus showed that the blood serum of patients with Burkitt's lymphoma and nasopharyngeal carcinoma and the serum of *Papio hamadryas* with a high titer of antibodies to EB virus precipitated approximately 70% radioactive material. The findings indicated that the virus contained in the ChSL-1 culture cells was similar to or identical with EBV.

4490 THE STRUCTURE AND BIOLOGICAL PROPERTIES OF HERPES SIMPLEX VIRUS DNA. (Eng.) Wilkie, N. M.; Clements, J. B.; Macnab, J. C. M.; Subak-Sharpe, J. H. *In: Tumor Viruses. Cold Spring Harbor Symposia on Quantitative Biology.* Cold Spring Harbor Lab. (Cold Spring Harbor, NY): vol. 39, pp. 657-666; 1975.

Escherichia coli and *Hemophilus influenzae* endonucleases were applied to the study of various herpesvirus DNA's. The distribution and nature of single-strand interruptions after subjecting the DNA to varying degrees of stress were determined by electrophoresis. Herpes simplex virus type 1 (HSV-1) broke down to an apparently random assortment of fragments, suggesting the absence of preferred shear sites. The calcium phosphate precipitation technique was extended to baby hamster kidney (BHK) cells and to the DNAs of several other herpesvirus species. Virions produced after infection with DNA from virions of different genotypes were identical to the original phenotype. Shearing abolished the plaque-producing ability. Some marker genes could be recovered from sheared fragments. DNA (intact or sheared) from temperature-sensitive and permissive HSV types 1 and 2 was used to infect primary rat embryo cell cultures. In most cases, infection with intact DNA led to plaque formation at the appropriate temperatures. With the type 1 mutants ts F, ts G, and ts I and the type 2 mutants ts 1 and ts 3, transformed foci were seen at 38 C. Transformation is clearly independent of the conditional lethal function of these mutants. Since fragments and conditional lethals can transform, it seems unlikely that the whole genome of herpes simplex virus is necessary for successful transformation. (30 references)

4491 HERPES SIMPLEX VIRUS DNA IN TRANSFORMED CELLS: SEQUENCE COMPLEXITY IN FIVE HAMSTER CELL LINES AND ONE DERIVED HAMSTER TUMOR. (Eng.) Frenkel, N. (Div. Biological Sciences, Univ. Chicago, Chicago, IL 60637); Locker, H,; Cox, B.; Roizman, B.; Rapp, F. *J Virol* 18(3): 885-893; 1976.

Hybridization analyses were conducted on DNA from five hamster embryo cell lines transformed by UV irradiation and labeled herpes simplex virus 2 (HSV-2) DNA. The cell lines used were 333-8-9, 333-2-29, 333-2-26, MS4-1, and Col 6-1. Viral DNA sequences were detected in all five cell lines tested, but none of the lines contained the full complement of HSV-2 DNA. The amount of viral DNA present in cells varied in different transformed cell lines and ranged from 8% to 32% of the HSV-2 DNA genome in one to three copies per cell. Two parallel passages of the same cell line (333-2-29) differed in the amount of viral DNA they contained. The authors also compared the viral DNA sequences present in one transformed cell line (333-8-9) propagated serially in culture for 80 passages, a tumor produced by inoculation of a newborn hamster with the 333-8-9 cells, and a cell line derived from a hamster tumor as above and propagated in culture for 32 passages. The results show that viral DNA present in the hamster tumor and in the cells derived from the tumor had a lower sequence complexity than that present in the original serially passaged 333-8-9 cell line.

4492 INTEGRATION OF SUGAR TRANSPORT AND METABOLISM IN VIRUS-TRANSFORMED CELLS IN CULTURE (Meeting Abstract). (Eng.) Bissell, M. J. (Lab. Chemical Biodynamics, Lawrence Berkeley Lab., Univ. California, Berkeley, CA 94720); Hatie, C. *J Cell Biol* 70(2/Part 2): 380a; 1976.

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See also:

* (Rev): 4220, 4221, 4222, 4223, 4224, 4225, 4226, 4227, 4228, 4229, 4230, 4231, 4232, 4233, 4234, 4237, 4241, 4255, 4285, 4286, 4287
* (Phys): 4416
* (Immun): 4557, 4567, 4572, 4574, 4577, 4580, 4581, 4602
* (Path): 4611, 4634, 4661

4557 LACK OF CORRELATION BETWEEN AGGLUTINABILITY, THE SURFACE DISTRIBUTION OF CON A AND POST-CONFLUENCE INHIBITION OF CELL DIVISION IN TEN CELL LINES. (Eng.) Ukena, T. E. (Dept. Pathology, Harvard Medical Sch., Boston, MA 02115); Goldman, E.; Benjamin, T. L.; Karnovsky, M. J. *Cell* 7(2): 213-222; 1976.

Agglutinability by concanavalin A, distribution of surface-bound concanavalin A, and maximal cell density in monolayer culture were examined under similar conditions in parallel cultures of ten established cell lines (3T3-Cl-1, 3T3-Cl-2, 3T3-Py-6, 3T3-Py-6-R1, SVT2, UCl-B, BHK-21, Py-BHK, WI-ICI, IC-21). The degree of agglutinability of the cell lines did not correlate with the presence or absence of patching of concanavalin A bound to the cell surface, as determined with a hemocyanin marker. Agglutinability was also not always correlated with the loss of post-confluence inhibition of cell division. Two clones of mouse 3T3 fibroblasts that maintained post-confluence inhibition of cell division and low agglutinability differed substantially with respect to the surface distribution of concanavalin A. Patching of concanavalin A binding sites is neither necessary nor sufficient to explain differences in agglutinability between cell lines.

4558 FACTORS INVOLVED IN CONCANAVALIN A AGGLUTINATION OF KB AND NC37 CELLS GROWN IN SUSPENSION CULTURE. (Eng.) Altenburg, B. (Dept. Virology and Epidemiology, Baylor Coll. Medicine, Houston, TX 77025); Steiner, S. *J Natl Cancer Inst* 56(1): 197-200; 1976.

NC37 and KB cells grown in suspension culture remained agglutinable at 23 C by concanavalin A (Con A) after sufficient glutaraldehyde fixation to prevent lateral mobility (clustering) of Con A binding sites. In contrast, at 4 C agglutination of the fixed cells was blocked. No significant differences in ^{3}H-Con A binding were observed between unfixed cells and glutaraldehyde-fixed cells at 23 C and unfixed cells at 4 C. Con A bound to fixed cells at 4 C produced agglutination when the cells were washed and then warmed to 23 C. Thus a cold-sensitive factor unrelated to the binding of Con A or the clustering of Con A sites is necessary for agglutination to occur.

4559 LIGHT AND ELECTRON MICROSCOPIC STUDIES OF THE ARRANGEMENT OF CONCANAVALIN A RECEPTORS ON THE TUMOR CELL SURFACE. (Ger.) Roth, J. (Pathologisches Institut, Friedrich-Schiller-Universitaet Jena, Jena, East Germany). *Wiss Z Friedrich Schiller Univ Jena Math Naturwiss Reihe* 24(2/3): 337-340; 1975.

Experiments designed to clarify the poor correlation between the number of lectin-binding sites per cell surface unit and the extent of lectin-induced agglutination are described. Ehrlich ascites carcinoma (EAC) cells were used with concanavalin A and ferritin as the label of choice, and freeze-etching was the preparatory technic of choice. Ferritin particles were arranged on the cell surface in foci of various sizes, between which nonlabeled surface areas were found. The configuration of Con A-ferritin particles did not corresond to the distribution of particles on the positive line of cleavage of the plasma membrane. This result is contrary to the 1:1 correspondence between particles at the cleavage and surface glycoproteins in phytohemagglutinin and blood group-A antigen experiments. The result is confirmed by unpublished studies showing no numerical correlation between intramembrane particles and Con A receptors on rabbit erythrocyte surfaces. It follows that not all particles representing intrinsic proteins of the plasma membrane are glycoprotein carriers. (23 references)

4560 THE TUMOR CELL PERIPHERY: GLYCOPROTEIN LECTIN RECEPTORS. (Eng.) Walborg, E. F., Jr.; Davis, E. M.; Gilliam, E. B.; Smith, D. F.; Neri, G. *In: Cellular Membranes and Tumor Cell Behavior. Twenty-Eighth Annual Symposium on Fundamental Cancer Research, 1975.* (Baltimore: Williams and Wilkins Co.): pp. 337-360; 1975.

Investigations on the lectin-induced agglutination of Novikoff hepatoma cells and the use of lectins as molecular probes to detect structural and/or topological alterations of plasma membrane glycoproteins associated with neoplastic transformation are reported. The Novikoff ascites hepatoma was maintained in 6- to 9-wk-old female Sprague-Dawley rats by weekly transplantation. Lectin-induced (concanavalin A, Con A; wheat germ agglutinin, WGA; and *Ricinus communis* agglutinin I and II, RCA I and RCA II) cytoagglutination assays were performed on intact and papain-treated cells. Papain digestion of Novikoff cells reduced the number of cell-surface Con A receptors by 73% and released glycopeptides from the cell surface. These glycopeptides possessed receptor activity for Con A, RCA I, and WGA. Novikoff cells remained agglutinable by all three lectins following papain digestion, indicating that there are two qualitatively distinct lectin receptors at the cell periphery, glycopeptide lectin receptors that can be cleaved from the cell surface by protease and protease-stable receptors. Gel filtration of the cell surface glycopeptides yielded a glycopeptide fraction that contained all the detectable RCA I and WGA receptor activities and over 80% of the Con A receptor activity. Further purification yielded a fraction, DAI-2,3, that represented only 4.3% of the weight of the glycopeptides cleaved from the cell surface but contained 48% of the Con A and 61% of the RCA I receptor activities. DAI-2,3 consisted of peptide chains that are highly substituted with oligosaccharide moieties. A study of glutaraldehyde fixation effects on the lectin-induced agglutination of Novikoff cells indicated that the minimum lectin density requirements for cytoagglutination are met in different ways for Con A- and WGA-induced agglutination. Inhibition of WGA-induced cytoagglutination by glutaraldehyde fixation indicated that lectin-induced lateral mobility of WGA receptors is required for maximal agglutination of Novikoff cells by WGA. Glutaraldehyde fixation did not inhibit Con A-induced cytoagglutination, indicating that lateral mobility of Con A receptors is not required for Con A-induced cytoagglutination and suggesting that the inherent mber and distribution of Con A receptors provide e minimum Con A receptor density required for toagglutination. (70 references)

61 SEPARATION OF POPULATIONS OF SENSITIZED LYMPHOID CELLS INTO FRACTIONS INHIBITING D FRACTIONS ENHANCING SYNGENEIC TUMOR GROWTH IN VO. (Eng.) Small, M. (Dept. Cell Biology, Weizmann ist. Science, Rehovot, Israel); Trainin, N. *J Immol* 117(1): 292-297; 1976.

ibpopulations of lymphoid cells sensitized against syngeneic tumor were separated by velocity sedimenation, and the fractions were injected together ith tumor cells into syngeneic hosts. Inbred C57BL/6 d C3H/eb mice 2 to 3 mo old were used. Tumor growth s assayed in males, and tumors were also transerred in males. The spleen cells for in vitro senitization were from females. The test tumors were a ibrosarcoma induced in C57BL mice by benzpyrene, a ibrosarcoma induced in C3H mice by methylcholanhrene, and a spontaneously occurring carcinoma of 57BL mice, all of which had been passaged in syneneic mice for more than a year. Spleen cells from umor-bearing mice or spleen cells sensitized on umor cell monolayers could be fractionated into a ubpopulation capable of inhibiting growth of the ame tumor and a separable fraction that enhanced rowth. The activity of the tumor-inhibiting lymphoytes was not apparent in the presence of the tumor-nhancing cells. The former activity was associated ith small lymphocytes and the latter with injection f larger cells. Preliminary experiments investigated he developmental relation between the cells responible for these opposing activities. The results uggest that cells in the tumor-inhibiting fraction re derived with the passage of time from cells in he tumor-enhancing fraction. (23 references)

562 ASSOCIATION BETWEEN TUMOR IMMUNOLOGY, STROMAL REACTION, AND TUMOR ENDOCARDITIS. Ger.) Stosiek, P. (Pathologisches Institut des ezirkskrankenhauses, DDR-8900 Gorlitz, East Germany). *irchows Arch (Pathol Anat)* 369(2): 143-153; 1975.

he stroma infiltration by neutrophils, lymphocytes, lasma cells, and macrophages and the extent of tromal fibrosis or necrosis were examined histologically in 124 tumors associated with endocarditis. he predominant localizations included 26 stomach, 17 pancreas, 16 biliary tract, 14 uterus, and 12 ung. The overwhelming tumor type was adenocarcinoma; here were no anaplastic forms. For comparison, 50 undifferentiated malignancies without endocarditis were studied, including 12 bronchial, 7 cervical, and 5 mammary tumors and 5 melanomas. In the endocarditis group only, neutrophil infiltration was marked and the fibrosis was the terminal type. The degree of necrosis differed little between the two groups. The endocarditis-adenocarcinoma group was also more reactive immunologically: tumor-specific antibodies (IgG and IgM) occurred in more than half the cases. In the undifferentiated tumor group tumor antigens were infrequent and complement-fixation was absent. (58 references)

4563 IMMUNOLOGY OF MALIGNANT MELANOMA. (Ger.) Schieferstein, G. (Univ.-Hautklinik, Liebermeisterstrasse 25, 74 Tubingen, West Germany). *Fortschr Med* 94(14): 833-837; 1976.

The immune reactions of Syrian hamsters, BALB/c mice, Marshall rats, and BD IX-rats, all females, to M Mel-1 hamster melanoma ascites were studied in vivo in an intraperitoneal diffusion chamber. The tumor cell doubling time varied, depending on initial peritoneal fluid composition; karyotyping and impulse cytophotometry showed a nearly equivalent distribution of proliferating cells at different times. Weekly ip or sc administration of increasing doses of melanoma cells, hamster lymphocytes, or hamster liver cells elicited rapid cell lysis 4 hr postimplantation, with ascites cell losses up to 10%. Cell recovery began slowly at about the sixth day postimplantation and continued to increase gradually, despite relatively high levels of serum cytotoxicity. The in vivo diffusion chamber method is recommended not only for a more facile interpretation of humoral response to tumor cell challenge but also for more direct assay of the effect of different concentrations of cytostatic agents, regardless of mode of introduction. (8 references)

4564 MITOGENIC ACTIVITY OF MELANOMA EXTRACTS IN LEUKOCYTE CULTURES. (Ger.) Lischka, G. (Fakultaet fuer klinische Medizin Mannheim, Universitaet Heidelberg, D-6800 Mannheim 1, Theodor-Kutzer-Ufer, West Germany). *Arch Dermatol Forsch* 253(2): 105-112; 1975.

An extract was prepared from primary melanomas to test its effect on the leukocyte cultures of eight melanoma patients at different metastatic levels and seven control subjects. The transformation index, or number of transformed cells per 1,000, varied insignificantly between experimental and control cultures, both of which were positively mitogenic to the extract. The effect of a homologous skin extract prepared from a normal subject was negative in both melanoma and control cultures. The mitogenic factor, which probably derives from the cytoplasm, cannot be a histocompatibility antigen. (34 references)

4565 VERTICAL TRANSMISSION OF TUMOUR RESISTANCE IN GUINEA PIGS. (Eng.) Wainberg, M. A. (Jewish General Hosp., Montreal, Canada); Minden, P.; Weiss, D. W. *Nature* 259(5540): 213-215; 1976.

Because vertical transmission of resistance to transplantable line 10 hepatocarcinoma was previously found in female guinea pigs treated with the supernatant of sonically disrupted BCG (BCG-SS), experiments were conducted to determine if this was also the case in guinea pigs treated with the methanol extraction residue (MER) fraction of BCG. Eight female strain 2 guinea pigs treated with single doses of MER (0.5-1.0 mg) and one guinea pig treated with BCG-SS (50 µg N) before or after id tumor cell implantation were resistant to a further challenge with 10^7cells. These animals were mated with normal males at least 60 days after the initial tumor cell challenge, and the resulting offspring were inoculated id with 10^6 tumor cells. Cf a total of 26 examined offspring, 9 of MER-treated mothers and 2 of the BCG-SS-treated mother exhibited complete tumor regression by about 7 days after challenge. Nine other animals exhibited a considerable delay in onset of death. Rapid tumor growth at the site of initial challenge, followed by death, was observed in each of 19 age-matched controls. Three of eight third-generation offspring survived tumor challenge as opposed to none of the six age-matched controls. Experiments in which the newborns of each of three MER-immunotherapy-cured females and three female controls were interchanged indicated that the vertical transmission observed is not related to factors transmitted through the milk. Tests for delayed cutaneous hypersensitivity did not reveal evidence of cellular antitumor immune activity in the offspring of tumor-immune mothers, before skin challenge with line 10 hepatocarcinoma cells.

4566 SENSITISATION OF NEUROBLASTOMA PATIENTS AND RELATED AND UNRELATED CONTACTS TO NEUROBLASTOMA EXTRACTS.

(Eng.) Graham-Pole, J. (Royal Hosp. Sick Children, Glasgow, Scotland); Ross, C. E.; Ogg, L. J.; Cochran, A. J. *Lancet* 1(7974): 1376-1379; 1976.

Sensitization to neuroblastoma extracts was tested by the leukocyte migration inhibition technique in 8 neuroblastoma patients aged 3 mo to 10 yr, 22 relatives, and 49 unrelated contacts. All three groups showed a high frequency of positive reactions compared with 61 controls having no exposure to neuroblastoma. The frequency of sensitization could be related to closeness of contact, but the nature of the sensitizing material and its mode of transmission are unknown. Virus studies by the sensitive techniques of tumor virology are suggested. The absence of case clustering and the low familial incidence of neuroblastoma indicate that simple exposure to the tumor is not a major oncogenic factor. (19 references)

4567 IMMUNE RESPONSE IN THE MOUSE TO ENDOGENOUS LEUKEMIA VIRUSES.

(Eng.) Nowinski, R. C.; Kaehler, S. L.; Burgess, R. R. *In: Tumor Viruses. Cold Spring Harbor Symposia on Quantitative Biology.* Cold Spring Harbor Lab. (Cold Spring Harbor, NY): vol. 39, pp. 1123-1128; 1975.

A sensitive radioimmune precipitation assay was used to detect immune responsiveness to endogenous leukemia virus (MuLV) in several inbred strains of laboratory mice. Antibody to MuLV was detected in the mice of almost all strains examined. Mice of some strains (I, SJL/J, NAB, and NZW) produced high titers of antibody, while mice of other strains showed either intermediate titers (DBA/2, C3H/An, HSFS, 129, GR, and AKR) or relatively low titers (c58, C57L/J, and PL/J). In general, older mice showed higher antibody titers. The mouse anti-MuLV antibodies were distributed among all four classes of immunoglobulins, but the highest titer antibodies were generally of the αG_1 and γG_2 classes. Immune precipitates formed between virus and natural mouse antibody were examined by polyacrylamide gel electrophoresis. The mouse antibodies were shown to be directed against four of the six major MuLV structural proteins: gp70, gp45, p15, and p10. Antibodies against p30 or p12 were never detected. Variations in the spectrum of the immune response and in the representation of antibodies in different immunoglobulin classes were observed in mice of different genotypes. For example, the γG_1 and γG_2 immunoglobulins of an NZB serum precipitated gp45 and p10, whereas the M immunoglobulins from the same serum precipitated gp45, p15, and p10; all antibodies in the serum of a NZW mouse were directed against gp70, gp45, and p15. (19 references)

4568 TUMORIGENICITY AND THE EXPRESSION OF CELL-SURFACE CARBOHYDRATES.

(Eng.) Killion, J. J. (Univ. Oklahoma Health Sciences Center, Oklahoma City, OK 73104); Wallenbrock, M. A.; Rogers, J. A., III; Kollmorgen, G. M.; Sansing, W. A.; Cantrell, J. L. *Nature* 261(5555): 54-56; 1976.

The tumorigenicity and elution profile on Con A-affinity columns of the transplantable mammary adenocarcinoma A-10 grown in vivo and in vitro were investigated. A-10 was maintained as an ascitic tumor in $B6AF_1$ (C57BL/6 x A/J) mice and in monolayer culture. An ip inoculum of 10^2 or 10^3 cells grown in vivo routinely caused 100% mortality in $B6AF_1$ mice, whereas similar inocula of cells grown in vitro caused about 50% mortality. Tumors grown in vivo contained a large subpopulation of cells eluted with phosphate-buffered saline (PBS). In contrast, cultured A-10 cells contained virtually no PBS-eluted subpopulation and were characterized by subpopulations released with different sugar solutions (α-methyl-D-mannoside, α-methyl-D-glucoside, sucrose, and α-D-glucose). After the first passage of cultured cells in normal mice, the cells eluted with PBS, and large numbers of the cells were released by sugar solutions. The tumorigenicity of these cells was comparable with that observed for cultured cells. After the second passage, however, the elution profile of the cells had reverted almost completely to that of tumor grown in vivo. Thus, the tumorigenicity of murine adenocarcinoma varies as a function of the tumor subpopulations present, as defined by their expression of cell-surface carbohydrates.

4569 IMMUNOLOGICAL CLASSIFICATION OF ACUTE LYMPHOBLASTIC LEUKAEMIAS: EVALUATION OF ITS CLINICAL SIGNIFICANCE IN A HUNDRED PATIENTS.

(Eng.) Brouet, J.-C. (Hopital Saint-Louis, 75475 Paris Cedex 10, France); Valensi, F.; Daniel, M.-T.; Flandrin, G.; Preud'homme, J.-L.; Seligmann, M. *Br J Haematol* 33(3): 319-328; 1976.

The use of T and B lymphocyte markers and different antisera raised against malignant B cells and fetal thymocytes allowed the classification of 100 patients (63 children under 16 yr and 37 adults) with acute lymphoblastic leukemia (ALL) into three groups: (a) patients with non-T, non-B ALL whose cells were devoid of conventional B and T markers but characterized by a leukemia associated antigen (69 cases); (b) patients with T-derived ALL (28 cases); and (c) patients with ALL of B cell origin (3 cases). Hematological and clinical correlations showed that those patients with T-derived ALL tended to have a higher leukocyte count and acid phosphatase positivity of blast cells, a higher tumor incidence, and a thymic mass. Survival curves for the two main patient groups were similar at 36 mo, but meningeal relapses were more frequent in patients with T-derived ALL. The experiments tended to validate the proposed classification of ALL into three main groups: B-derived ALL, including leukemias with Burkitt's tumor cells; T-derived ALL; and non-T, non-B ALL, which are characterized by leukemia associated antigens. (31 references)

4570 THEORY OF ALLOGENEIC REACTIVITY AND ITS RELEVANCE TO THE T-CELL RESPONSE TO NORMAL AND ONCOGENIC CELLS. (Eng.) Lafferty, K. J. (Univ. Colorado Medical Center, Denver, CO). *Adv Pathobiol* 4: 86-97; 1976.

The nature of allogeneic interactions is discussed and their relationship to normal antigen induction is outlined. A model for allogeneic interactions is developed and discussed in terms of the phylogeny of these interactions. It is assumed that a self-marker arose early in evolutionary development to prevent interaction between cells of like genotype. Inflammatory reactions are initiated when the self-marker carried by a stimulator cell is modified by some foreign agent (antigen). These modified stimulator cells can then activate other blood cells and so set off the inflammatory response. Invertebrate alloreactivity occurs when blood cells carrying different self-markers interact. The resulting severe inflammatory response disrupts vascular junctions and is seen as a rejection phenomenon. Vertebrate inflammatory reactions have an analogous mechanism. One component of vertebrate allogeneic interactions, the activation of immature blood cells, is essentially the same as invertebrate alloreactivity. Vertebrate T cells may be activated by normal self-stimulation following modification of the self-marker by antigen. Vertebrate immunocytes may also be activated by allogeneic stimulation. The application of the theory to the transplantation problem and to the tumor situation is discussed. (9 references)

4571 HUMAN B-LYMPHOCYTE ANTIGENS EXPRESSED BY LYMPHOCYTIC AND MYELOCYTIC LEUKEMIA CELLS. I. DETECTION BY RABBIT ANTISERA. (Eng.) Billing, R. (Depts. Surgery and Medical Microbiology Immunology, Sch. Medicine, Univ. California, Los Angeles, CA 90024); Rafizadeh, B.; Drew, I.; Hartman, G.; Gale, R.; Terasaki, P. *J Exp Med* 144(1): 167-178; 1976.

Tests of rabbit antisera were made with the use of purified subpopulations of lymphocytes as targets and immunofluorescent staining to detect minor populations of positive cells. A previously uncharacterized human B lymphocyte antigen was detected by rabbit antisera raised to papain digests of spleen cell membranes. The unabsorbed sera reacted in both cytotoxicity and immunofluorescent tests with normal B lymphocytes and cultured B cell lines but not with normal T lymphocytes or cultured T cell lines, which could provide the basis for more sensitive diagnostic aids for lymphoproliferative diseases. The cytotoxicity titers against B cells were as high as 1:32,000, whereas the same sera undiluted were negative against T cells. By immunofluorescent staining 6%-14% of unfractionated normal lymphocytes and 48%-85% of B-rich lymphocyte preparations were positive. Normal peripheral blood granulocytes, platelets, erythrocytes, and phytohemagglutinin blasts were negative. The antisera reacted with the same high titers against leukemia cells from approximately 70% of patients with acute lymphocytic leukemia, acute myelocytic leukemia, and chronic myelocytic leukemia, and 7/8 cases of chronic lymphocytic leukemia. From absorption studies it appeared that the same antigen was being expressed by leukemia cells and normal B lymphocytes. By immunofluorescent staining, the anti-B cell antisera were able to detect positive leukemia cells in the bone marrow of advanced leukemia patients and to monitor the elimination of these cells after chemotherapy. Soluble B cell antigen was found in some leukemia and lymphoma patient sera but not in normal sera. (19 references)

4572 RESPONSE OF B AND T LYMPHOCYTES AND OTHER BLOOD LEUKOCYTES IN CHICKENS WITH MAREK'S DISEASE. (Eng.) Payne, L. N.; Powell, P. C.; Rennie, M. *In: Tumor Viruses. Cold Spring Harbor Symposia on Quantitative Biology.* Cold Spring Harbor Lab. (Cold Spring Harbor, NY): vol. 39, pp. 817-826; 1975.

The blood leukocyte response in Marek's disease (MD) was studied, along with the cellular composition of MD lymphomas and the effect of removal of the bursa and thymus. HPRS strain chickens 1-3 days old were inoculated with 400 or 1,000 PFU of the HPRS-16 strain of MD virus. The chicks inoculated at 3 days had been x-irradiated, bursectomized, thymectomized, and/or sham-operated at 1 day. Between 25 and 69 days after infection, citrated blood was collected for blood leukocyte counts, cell suspensions were examined for proportions of T and B cells, heparinized blood and lymphomas were collected for preparation of lymphocyte suspensions, and tissues were taken for histological examination. On the average, the lymphomas were composed of 75.6% T cells and 17.1% B cells, and in some cases the sum of B and T cells fell far short of 100%. The proportion of B and T cells was reduced and cells with neither B nor T markers (null cells) were increased in the bursectomized birds. The proportion of Ig cells remained constant at about 3.4%, most of the B cells in the lymphomas appearing to be relatively undifferentiated. B cell numbers were decreased by irradiation and bursectomy in uninfected birds but not in infected birds. Infection increased the number of B and T cells, the other treatments having no effect on T cell number. Infection increased total lymphocyte counts, and thymectomy in uninfected birds significantly reduced counts. Infection significantly decreased the numbers of monocytes, the counts being higher in bursectomized and thymectomized birds. Basophils and eosinophils were also decreased by infection, which had no effect on heterophils. The results indicate that the lymphocytosis and leukemia in MD are mainly due to an increase in T cells, although B cells may also be a factor. (42 references)

4573 HUMAN T LYMPHOCYTE COLONIES IN AGAR: A COMPARISON WITH OTHER T CELL ASSAYS IN HEALTHY SUBJECTS AND CANCER PATIENTS. (Eng.) Wilson, J. D. (Dept. Medicine, Sch. Medicine, Univ. Auckland, Auckland, New Zealand); Dalton, G. *Aust J Exp Biol Med Sci* 54(1): 27-34; 1976.

A slightly modified T cell colony was used to examine T cell colony formation from the peripheral blood cells of healthy subjects and patients with cancer, as a group frequently exhibiting depressed cell-mediated immunity. Human lymphocyte colonies with T cell characteristics grew in agar from repeated mitotic divisions with phytohemagglutinin (PHA) stimulation. The colonies comprised spheres of tightly packed cells with up to 500-1,000 blastlike cells in each colon. A total of 65% of cells from pooled colonies bound SRBC treated with a sulfhydryl reagent. Between 1,100 and 2,500 colonies/10^6 peripheral blood lymphocytes developed when cell donors were healthy, but lower numbers (350-1,000 colonies/10^6 lymphocytes) were detected in blood from cancer patients. Comparison with other nonspecific assays of cell-mediated immunity showed that, although 66% of cancer patients were anergic (to five recall antigens) and 78% exhibited depressed mitotic activity in standard cultures with low-dose PHA, 100% of these patients revealed T cell colony formation below normal. Further studies of T lymphocyte colony-forming cells in healthy people and in a number of disease states may significantly advance our understanding of cell-mediated immunity. (11 references)

4574 CHARACTERIZATION OF EFFECTOR LYMPHOCYTES ASSOCIATED WITH IMMUNITY TO MURINE SARCOMA VIRUS (MSV) INDUCED TUMORS. I. PHYSICAL PROPERTIES OF CYTOLYTIC T LYMPHOCYTES GENERATED IN VITRO AND OF THEIR IMMEDIATE PROGENITORS. (Eng.) Plata, F. (Laboratoire d'Immunologie des Tumeurs, Batiment Gustave Roussy, Hopital Cochin, 75014 Paris, France); MacDonald, H. R.; Engers, H. D. *J Immunol* 117(1): 52-58; 1976.

The murine sarcoma virus (MSV) tumor system was used to determine if cytolytic thymus-derived lymphocytes (CTL) generated in mixed leukocyte-tumor cultures (MLTC) showed variations in size as a function of time in culture. The findings were compared with those observed for mixed leukocyte culture (MLC) cells. CTL were generated in secondary MLTC with syngeneic RB1-5 tumor cells as stimulating cells and with responding spleen cells from regressor mice that had rejected an MSV-induced tumor. CTL precursor cells were found to be exclusively of thymic origin, and non-T cells were apparently not required for CTL generation. When the size variations of CTL from syngeneic MLTC were analyzed by velocity sedimentation, a transition from small precursor cells to larger effector cells was found to occur during the first 5 days in culture, followed by a shift toward small-sized cells. Furthermore, the CTL generated in syngeneic MLTC in the MSV tumor immune system exhibited fundamental similarities when compared with those obtained in allogeneic MLC's. (25 references)

4575 ERYTHROCYTE RECEPTORS AND THYMUS-ASSOCIATED ANTIGENS ON HUMAN THYMOCYTES, MITOGEN-INDUCED BLASTS, AND ACUTE LEUKEMIA BLASTS. (Eng.) Sen, L. (Medical Coll. Wisconsin, Milwaukee, WI 53233); Mills, B.; Borella, L. *Cancer Res* 36(7): 2436-2441; 1976.

Human thymus cells and blasts from some patients with acute lymphoblastic leukemia (ALL) express similar cell surface properties, suggesting that ALL originates in the thymus or that these properties of ALL blasts reflect changes occurring during blastogenesis. To test these possibilities, a study was made on whether mitogen-induced blasts would express erythrocyte (E) receptors and thymus antigens found on blasts from E+ALL and on normal human thymocytes. E rosettes of blood T lymphocytes dissociated at 37 C; in contrast, rosettes formed by E+ALL blasts, human thymocytes, and pokeweed mitogen-stimulated blasts were stable at this temperature. Blasts that formed stable rosettes did not have cytoplasmic Ig, indicating that they were T lymphoblasts. By immunofluorescence and a radiolabeled antibody assay, a thymus antigen(s) that was present on the membrane of E+ALL blasts and on normal thymocytes, but not on normal blood T-lymphocytes [TL-like antigen(s)] was demonstrated. This antigen was not expressed on blasts induced by mitogens, indicating that it is not required for the expression of E-receptors stable at 37 C. The results support the concept that E+ALL results from the malignant transformation of thymus cells. (25 references)

4576 HYBRID-VERSUS-PARENTAL STRAIN REACTION. II. COMPARATIVE MORTALITY IN PARENTAL STRAIN MICE OF DIFFERENT H-2 GENOTYPES. (Eng.) Uphoff, D. E. (Lab. Physiology, Natl. Cancer Inst., Natl. Insts. Health, Bethesda, MD 20014). *J Natl Cancer Inst* 56(1): 163-166; 1976.

Lethally irradiated parental strain mice were inoculated with marrow from reciprocal hybrid donors. Hybrid cells recognized the quantitatively greater antigenicity of parental strain recipients. The severity of the hybrid-vs-hybrid reactions was compared. There was a significantly less severe reaction when DBA/2 mice were inoculated with (DBA/2 female x C57BL male)F_1 marrow than with (C57BL x DBA/2)F_1 marrow, which indicated a maternal influence on antigen recognition. The reaction of (DBA/2 x C57BL)F_1 against DBA/2 recipients was significantly less severe than the DBA/2 reaction against (DBA/2 x C57BL)F_1 recipients. All reactions involving C57BL were equally severe and more lethal than those involving DBA/2 mice. The strength of these reactions was a function of the relative tissue antigenicity and responsiveness of the parental strains of the hybrid cross. Both the expression of tissue antigens and the ability to recognize and respond to them are influenced by the maternal strain of the hybrid cross.

4577 RADIOIMMUNOASSAYS FOR THE 70,000-MOLECULAR-WEIGHT GLYCOPROTEINS OF ENDOGENOUS MOUSE TYPE C VIRUSES: VIRAL ANTIGEN EXPRESSION IN NORMAL MOUSE TISSUES AND SERA. (Eng.) Hino, S. (Lab. RNA Tumor Viruses, Natl. Cancer Inst., Bethesda, MD 20014); Stephenson, J. R.; Aaronson, S. A. *J Virol* 18(3): 933-941; 1976.

The 70,000 molecular wt glycoproteins (gp70) were purified from three prototype viruses and competition radioimmunoassays for each were developed. With use of these immunoassays, the antigenic determinants of gp70s of different classes of endogenous virus, isolated from NIH Swiss, BALB/c, NZB, and AKR strains were readily discriminated. In contrast, viruses of the same class were indistinguishable. These findings further document the existence of three distinct endogenous viruses of mouse cells. The levels of type C viral gp70 were quantitated in the tissues and sera of several inbred strains. The pattern of immunological reactivity of the gp70 detected in serum was indistinguishable from that of the viral gp70 partially purified from tissues of the same strain. Moreover, in each case it was indistinguishable from that of a specific class of endogenous virus. In virus-negative tissues of BALB/c and NIH Swiss mice, the viral gp70 detected was representative of a class III endogenous virus whose 12,000 molecular wt polypeptide was also expressed by the same cells.

4578 IMMUNOLOGICAL CROSS-REACTIVITY OF ANTIBODIES TO A SYNTHETIC UNDECAPEPTIDE ANALOGOUS TO THE AMINO TERMINAL SEGMENT OF CARCINOEMBRYONIC ANTIGEN, WITH THE INTACT PROTEIN AND WITH HUMAN SERA. (Eng.) Arnon, R. (Weizmann Inst. Science, Rehovot, Israel); Bustin, M.; Calef, E.; Chaitchik, S.; Haimovich, J.; Novik, N.; Sela, M. *Proc Natl Acad Sci USA* 73(6): 2123-2127; 1976.

The synthesis, by the solid phase technique, of the 11-amino acid NH_2-terminal peptide of carcinoembryonic antigen (CEA) and the development of a sensitive immunoassay specific to it are reported. The synthetic CEA(1-11) peptide was attached by a water-soluble carbodiimide reagent to multichain poly(DL-alanine) as well as to bovine serum albumin. Both macromolecular conjugates produced anti-CEA(1-11) peptide antibodies in rabbits. The specificity of this immunological system and the cross-reactivity between the peptide and intact CEA were investigated by two methods: passive hemagglutination and modified bacteriophage inactivation. Hemagglutination experiments showed that both anti-CEA(1-11) and anti-CEA sera agglutinated CEA(1-11)-coated SRBC; these reactions were inhibited with CEA(1-11) peptide. In experiments with the chemically modified bacteriophage technique, CEA(1-11)-coated phage was efficiently inactivated with antisera against the CEA(1-11) conjugates, and this inactivation could be totally inhibited with the free peptide. The semipure CEA, but not the pure protein, could also inhibit the phage inactivation, even though less efficiently. The sera of some cancer patients were tested for their capacity to inhibit the inactivation of CEA(1-11)-coated phage by anti-CEA(1-11) antiserum. The results indicate that sera from a large proportion of patients with adenocarcinomas of the digestive tract, pancreas, and breast are capable of inhibiting the above inactivation, whereas most normal sera are not. (31 references)

4579 ANTIBODY TO CARCINOEMBRYONIC ANTIGEN IN HAMSTERS BEARING GW-39 HUMAN TUMORS. (Eng.) Primus, F. J. (Dept. Immunology, Hoffmann-La Roche Inc., Nutley, NJ 07110); Wang, R. H.; Cohen, E.; Hansen, H. S.; Goldenberg, D. M. *Cancer Res* 36(7): 2176-2181; 1976.

The appearance of anticarcinoembryonic antigen (anti-CEA) activity in the sera of male Syrian hamsters bearing im heterografts of GW-39, a CEA-producing human colonic tumor, was demonstrated by radioimmunoassay using radioiodinated CEA purified from GW-39. Column chromatography, sucrose gradient ultracentrifugation, and radioimmunoelectrophoresis of pooled sera from tumor-bearing hamsters showed that the CEA antibody was of the immunoglobulin M type. The CEA antibody in tumor sera contained two specificities that reacted with different determinants on the CEA, one with a blood group-related determinant, the other against a unique site on the CEA. Sequential analysis of individual tumor-bearing animals revealed that an increase in tumor growth was concomitant with the appearance of circulating CEA and highest antibody titer, suggesting that immunoglobulin M antibody, antigen, or complexes thereof influence tumor growth in this model. (26 references)

4580 LOCALIZATION AND DISTRIBUTION OF ACTIN FIBERS IN NORMAL, TRANSFORMED AND REVERTANT CELLS. (Eng.) Weber, K.; Lazarides, E.; Goldman, R. D.; Vogel, A.; Pollack, R. *In: Tumor Viruses. Cold Spring Harbor Symposia on Quantitative Biology.* Cold Spring Harbor Lab. (Cold Spring Harbor, NY): vol. 39, pp. 363-369; 1975.

An antibody against electrophoretically purified SDS-denatured actin from mouse 3T3 cells (SV40-transformed clone SV101) was used to determine the presence of actin in normal rat embryo cells, growth-controlled mouse 3T3 cells, clones of these lines transformed by SV40 or murine sarcoma virus, and revertant clones. Evidence suggests that the major SV101 protein species with an electrophoretic molecular wt of 45,000 is indeed actin. The antibody cross-reacts with actin from cells from a variety of different species. Both light microscopy and darkfield UV optics revealed a variety of fibers in most 3T3 cells; these fibers apparently contain actin. In the absence of any restriction on growth, normal, untransformed fibroblasts were found to contain ''actin cables.'' Transformed clones isolated after infection with SV40 or MSV showed a drastic loss of strong actin fibers. The two density-sensitive revertants F1SV and M22 showed a remarkable recovery of the cabling pattern originally present in 3T3 or Balb but lost upon viral transformation. However, the serum-sensitive revertant LS showed a pattern similar to that of SV101 and was therefore distinctly different from the other two revertants. Neither serum concentration nor, by implication, the level of cellular cyclic AMP seemed to have any marked effect on the expression of actin cables. These results suggest at least four different levels of cellular actin organization: thick actin bundles, thinner bundles or threads, shorter and thinner actin aggregates, and G actin or soluble actin. (21 references)

4581 SURFACE ANTIGENS ASSOCIATED WITH THE TRANSFORMATION OF 3T3 MOUSE FIBROBLASTS BY SIMIAN VIRUS 40 AND POLYOMA VIRUS ASSAYED BY ISOELECTRIC EQUILIBRIUM METHOD. (Eng.) Sherbet, G. V.; Lakshmi, M. S. *In: Molecular Base of Malignancy: New Clinical and Therapeutic Evidence.* Edited by E. Deutsch; K. Moser; H. Rainer; A. Stacher. (Stuttgart, Germany: George Thieme Publishers): pp. 5-9; 1976.

Viral transformation is accompanied by the appearance of new antigens in the cell surface--these transformation-associated surface antigens (TASA) appear to be nonspecific for the transforming virus but are coded by the host cell. The detection of TASA on simian virus 40 (SV40) transformed 3T3 mouse fibroblasts is reported; they are host-cell specific and appear to be formed de novo. Tests of the isoelectric equilibrium following transformation indicate that SV3T3 and Py3T3 cells (SV40- and polyoma-transformed 3T3 fibroblasts) share certain antigens. The numbers of TASA and 3T3 antibody binding sites were calculated for the two transformed cell lines. The numbers of TASA sites were comparable; however, the Py3T3 cells had fewer 3T3 specific antigen sites. This indicates that the SV40 and polyoma viruses affect different features of the transformed phenotype. Studies using trypsin demonstrated that the new antigen sites were not cryptic normal 3T3 antigens, and concanavalin A binding studies suggested that the TASA are formed de novo by the host cells. (13 references)

4582 RHEUMATOID FACTOR AND TUMOR-HOST INTERACTION. (Eng.) Twomey, J. J. (Veterans Admin. Hosp., 2002 Holcombe Blvd., Houston, TX 77211); Rossen, R. D.; Lewis, V. M.; Laughter, A. H.; Douglass, C. C. *Proc Natl Acad Sci USA* 73(6): 2106-2108; 1976.

Evidence that rheumatoid factor (RF) is readily detected in serum from patients with solid tumors and that conventional tumor therapy contributes to the occurrence of this antibody response is reported. In this survey for RF seropositivity on patients with neoplasms, an 85% rate of positive screening tests was recorded under certain circumstances. This high rate of RF seropositivity occurred after irradiation and/or chemotherapy of breast and lung cancers. Treated patients with breast cancers who had no evidence of residual tumor had an 89% positive RF rate. Conversely, the incidence of RF seropositivity was low among untreated patients with similar tumors and treated patients with glioblastomas or multiple myeloma. The administration of cytotoxic drugs (such as azathiprene) was not itself associated with RF production even in renal allograft recipients. The data indicate that RF production occurs frequently after the therapy of certain tumors and suggest that in these circumstances RF may be an expression of tumor-host interaction. (19 references)

4583 THE OCCURRENCE OF ANTIBODIES AGAINST SINGLE-STRANDED DNA IN THE SERA OF PATIENTS WITH ACUTE AND CHRONIC LEUKAEMIA. (Eng.) Izui, S. (Hopital Cantonal, Geneva, Switzerland); Lambert, P. H.; Carpentier, N.; Miescher, P. A. *Clin Exp Immunol* 24(3): 379-384; 1976.

One hundred and seventy-five sera from 33 patients with acute myeloid leukemia, 42 patients with chronic myeloid leukemia, and 12 patients with acute lymphatic leukemia were examined radioimmunologically for the presence of antibodies against single- and double-stranded DNA. The levels of single-stranded DNA binding activity were significantly higher in all three types of leukemia compared to those of healthy controls. In contrast, none of these sera exhibited a positive reaction with double-stranded DNA. In some cases the level of serum anti-DNA antibodies increased after decrease of the WBC count. The presence of anti-DNA antibodies in leukemic patients may have some biological significance; the possibility cannot be excluded that such antibodies could be induced by a viral oncogenic agent. (23 references)

4584 STUDIES OF RECEPTORS ON NORMAL AND MALIGNANT TISSUE FROM MAN. (Eng.) Humphrey, L. J. (Univ. Kansas Medical Center, Rainbow Blvd. at 39th, Kansas City, KS 66103). *J Surg Oncol* 8(2): 125-130; 1976.

The reaction of unsensitized RBC and antibody-sensitized RBC (EA) with sections from human tumors as well as pathologic and normal tissues was studied. Sections from 17 human cancers were tested against RBC from guinea pig, chicken, alligator, monkey, rat, and rabbit. Seven tumor tissues adsorbed guinea pig RBC, and 11 sections adsorbed rat RBC; only one section adsorbed guinea pig RBC, and the other RBC were not adsorbed. This receptor was also seen with normal human tissues. Sections of malignant tissue lysed monkey RBC and agglutinated rabbit RBC. Most malignant tissues gave positive reactions with EA (sensitized with anti-sheep erythrocyte sera from rabbit or mouse), while only a few normal tissues did. The spleen, liver, and kidney sections of a patient who died of sepsis reacted with EA.

4585 ISOLATION FROM PATIENTS WITH BREAST CANCER OF ANTIBODIES SPECIFIC FOR ANTIGENS ASSOCIATED WITH BREAST CANCER AND OTHER MALIGNANT DISEASES. (Eng.) Gorsky, Y. (Hadassah Medical Sch., Hebrew Univ., Jerusalem, Israel); Vanky, F.; Sulitzeanu, D. *Proc Natl Acad Sci USA* 73(6): 2101-2105; 1976.

Investigations aimed at demonstrating the existence of antibodies against antigens associated with breast cancer and other malignancies are reported. Antibodies were specifically purified, with the use of pleural fluid antigens entrapped in a polyacrylamide gel as immunoadsorbent, from sera of patients with breast cancer and from the pleural effusion of an individual patient. Sera obtained from patients with breast cancer 3-4 wk after mastectomy were used as the source of standard antibody. Sera for assay were obtained from patients with various stages of breast cancer, patients with other malignancies, and healthy women. The purified antibodies were radioiodinated and tested for their capacity to bind to the pleural fluid adsorbent. Binding of the radiolabeled antibodies was inhibited by many of the sera from women with breast cancer to a much greater extent than sera of healthy women. With purified antibodies originating from the serum, 52% of the breast cancer sera and 16% of sera from patients with other malignancies were more inhibitory than were 95% of the normal sera. In the tests with antibodies eluted from pleural fluid, 24% of the breast cancer sera and 25% of the sera from patients with other neoplastic diseases were more inhibitory than were 95% of the normal sera. It was concluded that sera of patients with breast cancer may contain antibodies against breast cancer antigens as well as antibodies against antigens that are found in patients with other neoplastic diseases or in normal individuals. Individual breast cancer sera may inhibit binding of the labeled antibodies against breast cancer owing to their content of antibodies and/or antigen(s). (10 references)

4586 IgE LEVELS IN SERA OF CANCER PATIENTS. (Eng.) Winters, W. D. (Dept. Surgery, UCLA Sch. Medicine, Los Angeles, CA 90024); Heiner, D. C. *J Allergy Clin Immunol* 57(2): 181-186; 1976.

Immunoglobulin E (IgE) was measured in the sera of 18 healthy adult volunteer donors, 67 adults with various types of solid neoplasms, and 17 adults with clinical allergy by means of a double-antibody radioimmunoassay. There was no significant difference in the geometric mean serum IgE level between all cancer subjects and the healthy control subjects except that cancer and noncancer patients who had definite clinical allergies showed an increased mean IgE level. Similarly, there was no significant difference in the mean IgE level of any of the six cancer subgroups studied when compared to the control mean. Thus, there was no evidence reflected in serum levels tha IgE plays a role in the immunopathology of the cancer population tested.

4587 IMMUNOLOGICAL AND BIOCHEMICAL COMPARISONS OF MEMBRANE COMPONENTS FROM NORMAL AND TRANSFORMED BALB/C CELLS (Meeting Abstract). (Eng.) Van Nest, G. A. (Univ. Arizona, Tucson, AZ 85724); Kamm, A. R.; Grimes, W. J. *Fed Proc* 35(7): 1682; 1976.

4588 IMMUNOCHEMICAL CHARACTERIZATION OF BLOCKING AND INHIBITING FACTORS ASSOCIATED WITH THE CANINE VENEREAL TUMOR (Meeting Abstract). (Eng.) Beschorner, W. E. (Veterans Admin. West Side Hosp., Chicago, IL); Hess, A. D.; Nerenberg, S. T.; Epstein, R. B. *Clin Res* 24(4): 602A; 1976.

4589 TRANSPLANTATION IMMUNOGENETICS OF THE CANINE TRANSMISSIBLE VENEREAL TUMOR (Meeting Abstract). (Eng.) Bennet, B. T. (Univ. Illinois at Medical Center, Chicago, IL 60616). *Diss Abstr Int* [*B*] 36(6): 2714-B; 1975.

4590 BCG-INDUCED SPECIFIC IMMUNITY TO CHEMICALLY INDUCED, HISTOCOMPATIBLE FIBROSARCOMA TRANSPLANT LINES IN CHICKENS (Meeting Abstract). (Eng.) Palladino, M. A. (N.Y.U. Sch. Medicine, New York, NY 10003); Lerman, S. P.; Thorbecke, G. J. *Clin Res* 24(4): 607A; 1976.

4591 ADJUVANT CONTACT SUPPRESSION OF RAT AND HUMAN TUMOURS IN ATHYMIC NUDE MICE (Meeting Abstract). (Eng.) Pimm, M. V. (Cancer Res. Campaign Lab., Univ. Nottingham, Nottingham, England). *Br J Cancer* 34(3): 317; 1976.

4592 TRANSFER OF HUMAN TUMOR-SPECIFIC IMMUNITY IN VITRO WITH RNA (Meeting Abstract). (Eng.) Wang, B. S. (Dept. Surgery, Boston Univ. Medical Center, Boston, MA 02215); Mannick, J. A.; Deckers, P. J. *Clin Res* 24(4): 608A; 1976.

4593 ENHANCEMENT OF THE IMMUNOLOGIC PROPERTIES OF NEOPLASTIC CELLS (Meeting Abstract). (Eng.) Hakim, A. A. (Depts. Physiology and Surgery, Univ. Illinois at Medical Center, Chicago, IL). *Clin Res* 24(4): 548A; 1976.

4594 MODULATION OF THE ONCOGENIC CHARACTERISTICS OF SPONTANEOUS MAMMARY ADENOCARCINOMA (Meeting Abstract). (Eng.) Hakim, A. A. (Depts. Surgery and Physiology, Univ. Illinois at Medical Center, Chicago, IL). *Clin Res* 24(4): 549A; 1976.

4595 IMMUNIZATION WITH SOLUBILIZED EXTRACTS OF RAT TUMOURS AND FRACTIONS OF TUMOUR-BEARER SERUM (Meeting Abstract). (Eng.) Price, M. R. (Cancer Res. Campaign Lab., Univ. Nottingham, Nottingham, England); Preston, V. E.; Zoeller, M. *Br J Cancer* 34(3): 316; 1976.

4596 LOSS OF RESISTANCE TO TUMOR GROWTH IN THE NZB/NZW LUPUS MODEL (Meeting Abstract). (Eng.) Branahl, J. (Univ. Missouri, Columbia, MO 65201); Morris, A. D.; Mierzwa, P. *Clin Res* 24(4): 542A; 1976.

4597 HUMAN ALPHA-FETOPROTEIN (HAFP): CORRELATION OF IMMUNOSUPPRESSIVE PROPERTIES WITH ELECTROPHORETIC VARIANTS (Meeting Abstract). (Eng.) Lester, E. P. (Dept. Medicine, Univ. Chicago, Chicago, IL 60637); Miller, J. B.; Yachnin, S. *Clin Res* 24(4): 586A; 1976.

4598 ALPHA-FETOPROTEIN (AFP) IN CANCER-PRONE FAMILIES (Meeting Abstract). (Eng.) Keller, R. H. (Mayo Medical and Graduate Sch., Rochester, MN); Guirgis, H.; Lynch, H.; Tomasi, T. B. *Clin Res* 24(3): 331A; 1976.

4599 PLASMA PROSTAGLANDIN LEVELS AND DELAYED HYPERSENSITIVITY IN CANCER PATIENTS (Meeting Abstract). (Eng.) Harvey, H. (Dept. Medicine, Milton S. Hershey Medical Center Pennsylvania State Univ., Hershey, PA); Allegra, J.; Luderer, J.; Brenner, D.; Trautlein, J.; Gillin, M.; White, D.; Demers, L.; Lipton, A. *Clin Res* 24(3): 376A; 1976.

4600 IMMUNOLOGIC DISORDERS AND MALIGNANCIES IN FIVE YOUNG BROTHERS (Meeting Abstract). (Eng.) Purtilo, D. (Dept. Pathology, Worcester City Hosp., Worcester, MA); Yang, J.; Riordan, J.; DeFlorio, D. *Clin Res* 24(3): 335A; 1976.

4601 INHIBITION OF CELL-MEDIATED IMMUNITY TO RAT SARCOMAS FOLLOWING TREATMENT WITH ISOLATED TUMOUR ANTIGEN PREPARATIONS (Meeting Abstract). (Eng.) Embleton, M. J. (Cancer Res. Campaign Lab., Univ. Nottingham, Nottingham, England). *Br J Cancer* 34(3): 316-317; 1976.

4602 THE ROLE OF MEMBRANE ASSOCIATION OF ANTIGENS IN INDUCTION OF CELL MEDIATED IMMUNITY TO VIRUSES (Meeting Abstract). (Eng.) Rytel, M. W. (Medical Coll. Wisconsin, Milwaukee, WI); Niebojewski, R.; Broor, S. *Clin Res* 24(4): 578A; 1976.

4603 ANTIGENIC CROSS-REACTIVITY IN PROSTATIC CANCER (Meeting Abstract). (Eng.) Bhatti, R. A. (Cook County Hosp., Chicago, IL); Ablin, R. J.; Bruns, G. R.; Guinan, P. D. *Clin Res* 24(4): 548A; 1976.

4604 BIPHASIC EFFECTS OF TUMOR ASSOCIATED ANTIGENS (TAA) ON HOST IMMUNE RESPONSE (IMR) (Meeting Abstract). (Eng.) Nordlund, J. (Dept. Dermatology and Pathology, West Haven Veterans Admin. Hosp., West Haven, CT); Cone, R.; Gershon, R. K.; Ackles, A. *Clin Res* 24(3): 495A; 1976.

4605 A PLASMA MEMBRANE TUMOR ASSOCIATED ANTIGEN PRESENT IN OAT CELL CARCINOMA OF THE LUNG AND UNDETECTABLE IN NORMAL ADULT TISSUE (Meeting Abstract). (Eng.) Bell, C. E. (Dept. Pathology, Washington Univ. Sch. Medicine, St. Louis, MO 63130); Seetharam, S. *Clin Res* 24(3): 374A; 1976.

4606 HUMAN LEUKEMIC NEOANTIGENS: CLINICAL SPECTRUM AND PREDICTIVE SIGNIFICANCE (Meeting Abstract). (Eng.) Pisciotta, A. V. (Blood Res. Lab., Medical Coll. Wisconsin, Milwaukee, WI); Wilson, B. R.; Doucette, S. *Clin Res* 24(4): 572A; 1976.

4607 ANOMALOUS HL-A REACTIVITY IN ACUTE LYMPHOCYTIC LEUKEMIA (ALL) (Meeting Abstract). (Eng.) Huntington, S. J. (State Univ. New York, Upstate Medical Center, Syracuse, NY 13210); Davey, F. R. *Fed Proc* 35(3): 414; 1976.

4608 DETERMINATION OF CELL CYCLE PHASE SPECIFIC SURFACE MARKERS IN ACUTE LYMPHOCYTIC LEUKEMIA (Meeting Abstract). (Eng.) Dow, L. W. (St. Jude Children's Res. Hosp., Memphis, TN); Mauer, A. M. *Clin Res* 24(3): 306A; 1976.

4609 EXPRESSION OF ANTIGEN MARKER OF HUMAN THYMOCYTES IN NORMAL AND LEUKEMIC CELLS (Meeting Abstract). (Eng.) Chechik, B. E. (Dept. Immunology, Hosp. for Sick Children, Toronto, Canada); Pyke, K. W.; Gelfand, E. W. *Clin Res* 24(3): 481A; 1976.

See also:

* (Rev): 4233, 4234, 4235, 4236, 4237, 4238, 4239, 4240, 4266, 4267, 4287, 4288, 4289, 4290, 4291
* (Chem): 4344, 4350
* (Phys): 4385, 4395, 4396
* (Viral): 4446, 4453, 4461, 4483, 4485, 4488, 4506, 4507, 4511, 4512, 4513, 4514, 4515, 4516, 4517, 4520, 4529, 4531, 4532, 4550, 4553, 4556
* (Path): 4616, 4646

4610 MONOCYTOPOIESIS IN CHRONIC ECZEMATOUS DISEASES, PSORIASIS VULGARIS, AND MYCOSIS FUNGOIDES. (Eng.) Meuret, G. (Medizinische Klinik C, Kantonsspital, 9006 St. Gallen, Switzerland); Schmitt, E.; Hagedorn, M. *J Invest Dermatol* 66(1): 22-28; 1976.

Monocytopoiesis and blood monocytes were examined in eight patients with disseminated chronic eczematous diseases, eight patients with disseminated psoriasis vulgaris, and eight patients with mycosis fungoides in plaque or tumor stage. Monocytopoiesis was moderately stimulated in all the patients. The stimulation manifested itself by: a rise in the relative number of promonocytes in bone marrow in all patients with eczema, in one out of eight patients with psoriasis, and in seven out of nine patients with mycosis fungoides; a rise in [^{3}H]thymidine labeling indices of medullar promonocytes (8/8 eczema, 7/7 psoriasis, 8/9 mycosis fungoides); and a rise in the naphthol-AS-D-chloroacetate esterase activity of blood monocytes, indicating premature monocyte marrow egress (3/5 eczema, 7/8 psoriasis, 9/9 mycosis fungoides). In eczema and psoriasis, the mean enhancement of monocytopoietic activity was similar (but less pronounced than in mycosis fungoides). The results could indicate an increased monocyte consumption by pathologic, immunologic, and/or inflammatory processes.

4611 LEUKEMIC MITOCHONDRIA. II. ACUTE MONOBLASTIC LEUKEMIA. (Eng.) Szekely, I. E. (Harrisburg Hosp. Inst. Pathology, South Front St., Harrisburg, PA 17101); Fischer, D. R.; Schumacher, H. R. *Cancer* 37(2): 805-811; 1976.

Qualitative and quantitative ultrastructural studies were performed on mitochondria of leukemic monoblasts from 15 patients with acute monoblastic leukemia. Similar comparative observations were made on mitochondria of myeloblasts from 14 hematologically normal controls. No significant quantitative differences were noted between normal and leukemic mitochondria. Area measurements were approximately equal. Qualitative differences between the two groups consisted of increased numbers of irregularly shaped mitochondria, damaged mitochondrial membranes, mitochondria with damaged matrix, and small granules in mitochondria in the leukemic group. Leukemic cells exhibited nuclear-mitochondrial contact and virus-like particles within damaged mitochondria. To confirm the presence of virus-like particles and to aid in the understanding of nuclear-mitochondrial interaction, a C-type virus producer Moloney sarcoma virus-Moloney leukemia virus-infected rat embryo cell culture was used for additional analysis. Mitochondrial abnormalities and increased frequency of virus in damaged mitochondria, often attached to mitochondrial membranes, were noted. Several lysosomes exhibited accumulations of virus and budding into lysosomes from lysosomal membranes. Mitochondria are important organelles in glycolytic-oxidative phosphorylation pathways, and carry extranuclear genetic information. Further studies of morphologic and biochemical abnormalities of leukemic mitochondria and the interaction between the mitochondria and the nuclei in leukemic cells are needed to provide researchers with data on extranuclear factors operating in oncogenesis.

4612 EOSINOPHILIA IN THE PRELEUKEMIC PHASE OF ACUTE MYELOID LEUKEMIA. (Ger.) Binder, C. (Universitaets-Kinderklinik Wien, Waehringer Guertel 74-76, A-1090 Vienna, Austria); Granditsch, G. *Paediatr Paedol* 11(1): 234-239; 1976.

The role of eosinophilia in the preleukemic processes of acute myeloid leukemia (AML) was studied in a 4.5-yr-old girl whose serum showed a leukocytosis of 25,000 WBC/mm^3 and an eosinophilia of 36% in differential hematology (otherwise, she was symptom-free). One year later a WBC of 6,700/mm^3 preceded a cerebral pseudotumor caused by meningosis that was treated with intrathecal methotrexate. The eosinophilic chemotactic factors seemed to be a late AML manifestation by the malignant cells present and seemed to have evolved from an intact immune system against these cells. The tumor cells showed no eosinophilia, and immunoreactions were failing. The findings of anemia (1.6 x 10^6 RBC/mm^3, HB 5.2 g%) and thrombocytopenia (59,000/mm^3) confirmed AML.

4613 ULCER AND CHRONIC MYELOID LEUKEMIA, AN ASSOCIATION OR COINCIDENCE IN THREE CASES. (Fre.) Puchelle, J. C. (Clinique Medicale ''A'', C.H.U. de Nancy-Brabois, France); Lederlin, P.; Guerci, O.; Aymard, J. P.; Witz, F. *Ann Med Nancy* 15: 57-58, 60; 1976.

Three cases of an association between chronic myeloid leukemia (CML) and gastric or duodenal ulcers are described. The first patient, a man of 51, who had received medical treatment for an ulcer 5 yr before, became ill with CML in 1972. He presented a typical ulcer syndrome that was eliminated. Therapy by mitomycin and Misulban induced a partial remission in the leukemia. Two years later, the patient complained of a recurrence of gastric distress. This led to the discovery of a large subcardial benign ulceration. Surgery did not seem advisable because of the associated CML. Classic medical treatment continued for the ulcer. The second patient, a man aged 50, had twice received medical treatment for an ulcer, 20 yr and 5 yr before the discovery of CML. Four months after the start of hemopathy, he developed a new ulcer with an aspect of intense gastritis but no signs of neoplasia. Medical treatment resulted in the disappearance of the symptoms while the CML remained controlled by hydroxyurea. The third patient, a man aged 34, presented concurrent symptoms of typical CML and gastric pain. Fibroscopy revealed a large pyloric ulcer, and antiulcer therapy was begun immediately. Because of the ulcer, no antimitotic agents could be used against the CML. Instead, the patient received splenic radiotherapy (1,100 rads), and a remission occurred in 3 wk. Since then, his condition has remained satisfactory. Of 81 CML cases on file, 9 (11%) were associated with ulcers. Eight occurred before the leukemia, one concurrently. An ulcerous condition may favor the appearance of leukemia, but no relationship of this sort has been demonstrated. The ulcer-leukemia association creates problems, as the treatment for hemopathy may awaken a dormant ulcer or delay the healing of a recurrence. Thus, the medical treatment for ulcers seems almost always preferable and the avoidance of surgery logical.

4614 THE PATHOLOGIC CHARACTERISTICS OF LEUKOSTASIS AND LEUKEMIC NODULES OCCURRING IN THE CENTRAL NERVOUS SYSTEM OF GUNIEA PIGS WITH L_2C/NB LEUKEMIA. (Eng.) Nadel, E. M. (Medical Univ. South Carolina, Charleston, SC); Nelson, J. S. *J Neuropathol Exp Neurol* 35(1): 75-89; 1976.

Leukostasis and leukemic nodules were found in the CNS of at least 75% of guinea pigs during the terminal stages of untreated L_2C/NB leukemia, a transmissible, acute lymphocytic leukemia. The CNS lesions developed after extensive visceral leukemic infiltration at a time when the WBC count was rising to 10^5-5 x 10^5 cells/mm^3, and the differential examination showed predominantly blasts. Leukostasis preceded formation of the nodule. Both lesions might be found in any part of the CNS (including the spinal cord) but were most numerous in the diencephalon and rostal brainstem. Ultrastructural studies demonstrated that parenchymal leukostasis developed chiefly within capillaries, and was associated with endothelial cell degeneration and necrosis. Fibrin deposits were not seen within affected capillaries. The sequence of development, pathologic and hematologic characteristics of leukostasis and leukemic nodules in human and L_2C/NB leukemia were virtually identical.

4615 IDENTICAL GENETIC BASIS FOR LYMPHOSARCOMA AND HEMOLYTIC ANEMIA IN THE RABBIT. (Eng.) Fox, R. R. (Jackson Lab., Bar Harbor, ME 04609); Meier, H. *J Hered* 67(2): 99-102; 1976.

WH/J and XJ rabbits, which are known to transmit lymphosarcoma (ls) and hemolytic anemia (ha), respectively, were crossbred and tested for allelism of the ls and ha genes. Results indicated that they were both allelic and identical by descent. The two different conditions resulted from an interaction of these genes with the host genotype. Hemolytic anemia was the primary cause of death in compound heterozygotes (ha/ls), with increasing lymphoproliferative disease with age. Ages of death of histologically confirmed rabbits ranged from 5 days to 22.5 mo, with an average of 10.5 mo. The symbol ha will now be used to represent the gene for either disorder.

4616 LEUKAEMIC LYMPHOSARCOMAS: RESPECTIVE PROGNOSIS OF THE THREE TYPES: PROLYMPHOCYTIC, LYMPHOBLASTIC (OR LYMPHOBLASTOID) AND IMMUNOBLASTIC. (Eng.) Mathe, G. (Institut de Cancerologie et d'Immunogenetique, Hopital Paul-Brousse, Villejuif, France); Belpomme, D.; Dantchev, D.; Pouillart, P.; Schlumberger, J. R.; Lafleur, M. *Blood Cells* 1(1): 25-36; 1975.

Forty cases of leukemic lymphosarcoma (LLS) were investigated by cytologic, histologic, electron microscopic, and cell surface marker (T and B) studies. Three types of LLS were cytologically distinguished; immunoblastic, lymphoblastic (or lymphoblastoid), and prolymphocytic LLS. They are either nodular or diffuse: the nodular variety may be lymphoblastic or prolymphocytic and its cells are B markers. The diffuse variety may be immunoblastic, lymphoblastic, or prolymphocytic and its cells possess T or B markers or none. Therapeutic responsiveness is greatest in the prolymphocytic type and least in the immunoblastic. Prognosis is best for the prolymphocytic and worst for the immunoblastic type. These data emphasize the diagnostic importance of cell typing in LLS, as determined by cytological examination of smears in conjunction with the other investigations mentioned.

4617 NON-HODGKIN'S LYMPHOMA IN CHILDREN. (Eng.) Wollner, N. (Memorial Sloan-Kettering Cancer Center, 1275 York Ave., New York, NY 10021). *Pediatr Clin North Am* 23(2): 371-378; 1976.

The incidence, histology, primary disease sites, staging, initial workup, symptomatology, and treatment of non-Hodgkin's lymphomas in childhood are discussed. Approximately 400 new cases appear yearly in children aged 15 yr or younger, although the real incidence is difficult to estimate. These tumors can be classified according to pattern, type, and degree of differentiation by Rappaport's system. Diffuse lymphocytic poorly differentiated, diffuse histiocytic, and diffuse undifferentiated tumors are most common in children. The most common sites in children are: (a) gastrointestinal tract with small or large bowel; (b) skeletal, ovarian, nasopharyngeal, sc, renal, liver, thyroid, and CNS. According to a proposed system of staging, Stage I involves one site, Stage II involves two or more sites above or below the diaphragm, Stage III involves extensive intrathoracic and extensive intra-abdominal sites, and Stage IV involves one of the above with bone marrow and/or CNS involvement. Different clinical and laboratory manifestions are associated with meningeal, focal, and mixed meningeal/focal disease. The initial workup should include analysis of the blood, urine, spinal fluid, and bone marrow, with radiologic studies. Symptoms may vary considerably according to the primary site. The most important prognostic factor in non-Hodgkin's lymphoma is early intensive treatment. A treatment protocol involving cyclophosphamide in a high dose push; radiation therapy to bulky sites; induction, consolidation, and maintenance; and maximum protection of the CNS has improved the survival rate (72%) in a group of 58 patients. (11 references)

4618 ROLE OF HEREDITARY FACTORS IN THE SPREAD OF BOVINE LEUKEMIA. (Rus.) Khavruk, A. F. (Cherkassk State Experimental Agricultural Station, Cherkassk, USSR); Kuts, P. I.; Arkusha, N. S. *Veterinariia* (2): 49-56; 1976.

Three investigations have revealed the essential role of genetic factors in the spread of bovine leukemia. In the first, analysis of the morbidity of the disease in mothers and their offspring clearly indicated a vertical distribution of the predisposition for bovine leukemia. A study of the linear and familial traits of a second group of diseased cattle showed that recessive pathological genes were much more likely to pass from a heterozygous to a homozygous state than other diseased genes, the probability increasing with the mating of parents from leukemic families. The theory that bovine leukemia is transmitted only by the mother was not confirmed. A third study traced bovine leukemia in 59/82 affected cattle to the same diseased bull and indicated that any degree of inbreeding increases the possibility of inheriting the disease. The use of animals free of familial leukemia is crucial for the reproduction of healthy herds. Only isolated outbreaks of the disease can be managed successfully within the herd.

4619 NON-RANDOM AND RANDOM CHROMOSOMAL ABNORMALITIES IN TRANSFORMED CHRONIC GRANULOCYTIC LEUKAEMIA. (Eng.) Sharp, J. C. (Children's Hosp., Sheffield, England); Potter, A. M.; Wood, J. K. *Scand J Haematol* 16(1): 5-12; 1976.

Chromosome abnormalities were investigated in 10 consecutive cases of transformed chronic granulocytic leukemia. Bone marrow and peripheral blood samples were harvested for chromosome studies by an adaptation of the direct method. Chromosome abnormalities were identified with use of a banding technique. All 10 cases had chromosomal abnormalities in addition to the basic translocation (Philadelphia chromosome, Ph), a situation which contrasts with acute myeloblastic leukemia de novo, where 50% of cases may have abnormalities. Seven cases had nonrandom changes, trisomy 8 being a constant feature associated in each case either with a 17 abnormality or a second Ph or both (or, in some cases, with other abnormalities). In two cases serial studies were performed and additional abnormalities were found; these antedated transformation by 1 wk and 2 mo, respectively and suggested that transformation in chronic granulocytic leukemia might be preceded by additional chromosome abnormalities. Previous studies indicated that, apart from numerical changes in the Y, additional chromosome abnormalities do not occur in the chronic phase of granulocytic leukemia, but all the transformed cases in this series had such karyotypes. To overcome the difficulties in relating additional chromosome abnormalities to transformation, careful monitoring of the patient throughout the disease is required. (10 references)

4620 INCIDENCE AND CLINICAL FEATURES OF PITUITARY TUMORS IN CASES OF CENTRAL CUSHING'S SYNDROME. (Ger.) Barwich, D. (Medizinische Universitaets Polyklinik, Luisenstrasse 5, D-6900 Heidelberg, West Germany). *Acta Neurochir (Wien)* 33(3/4): 241-247; 1976.

A case history of pituitary tumorigenesis following adrenalectomy for management of Cushing's syndrome is described. A 28-yr-old female presented with adrenocortical hyperplasia. Ten months after total adrenalectomy and cortisone substitution therapy (37.5 mg daily), the skin, vermilion borders, and mucosa became rapidly overpigmented and resembled Addison's disease. Radiologically, a balloon-shaped shadow appeared in the sella postoperatively, along with severe headaches, visual deficit, amenorrhea, and loss of consciousness. Total bilateral ophthalmoplegia and approximately total amaurosis were found. Intervention included partial ablation of an extensive infra-, para- and suprasellar growth. A necrotic, eosinophilic adenoma was confirmed histologically. These adenomas are of the Nelson type (nodular hyperplasia), in which hypothalamic suppression of the corticotropin-releasing factor fails, and there is a surfeit of serum ACTH in adrenalectomized patients receiving hormonal substitution. Oral dexamethasone (5 mg five times daily) was transiently successful in managing the excess ACTH. (10 references)

4621 PITUITARY TUMORS AND HYPERPROLACTINEMIA. (Eng.) Malarkey, W. B. (Ohio State Univ. Hosp., 410 W. Tenth Ave., Columbus, OH 43210); Johnson, J. C. *Arch Intern Med* 136(1): 40-44; 1976.

In order to determine if the degree of serum prolactin elevation or growth hormone depression in patients with hyperprolactinemia reflects the amount of neoplastic tissue present, secretory patterns and 24-hr mean concentrations of serum prolactin and growth hormone were determined in nine patients with increased pituitary volume. Hyperprolactinemia was demonstrated in eight of the nine patients with clinical evidence of pituitary tumors without acromegaly or Cushing syndrome. Hourly sampling for 24 hr disclosed elevation of serum prolactin concentrations, whereas measurable serum growth hormone levels were found rarely. Although eight patients were hypersecreting prolactin, only four were lactating. Prolactin secretion was characterized by moderate hourly fluctuations of serum levels and absence or blunting of the normal sleep-related augmentation of secretion. Patients with the highest serum prolactin concentrations tended to have the largest pituitary tumors as indicated by pneumoencephalography. In two patients followed-up with serum prolactin determinations after treatment, a fall in serum prolactin concentrations correlated with clinical improvement. Future study will hopefully establish the value of serum prolactin determinations in following tumor growth before and after pituitary ablative therapy.

4622 PLASMA AND PARATHYROID TUMOR TISSUE GASTRIN AND HYPERPARATHYROIDISM. (Eng.) Wesdorp, R. I. C. (Massachusetts General Hosp., 32 Fruit St., Boston, MA 02114); Wang, C. A.; Hirsch, H.; Fischer*, J. E. *Am J Surg* 131(1): 60-63; 1976.

Hyperparathyroidism has been associated with an increased incidence of duodenal ulcer, increased acid secretion, and increased plasma gastrin levels. A relationship between these changes, increased serum calcium levels, and the increased incidence of peptic ulceration has been suggested, (especially since increased plasma gastrin levels, serum calcium levels, and gastric acid secretion decrease after parathyroidectomy). A study was made of 15 patients with primary hyperparathyroidism, plasma gastrin and serum calcium levels before and after parathyroidectomy, as well as the gastrin content of parathyroid tumor tissue. The mean basal plasma gastrin level before operation was significantly greater than that of a control group and decreased significantly after operation, in contrast to serum calcium levels. No positive correlation could be found between plasma gastrin and serum calcium levels before and after operation. Parathyroid tumor tissue was assayed for gastrin content by radioimmunoassay, and no detectable amounts of gastrin could be recovered from any tumor. The results did not support the concept that the extragastric source of gastrin in patients with hyperparathyroidism was the parathyroid adenoma.

4623 FAMILIAL HYPERPARATHYROIDISM: DESCRIPTION OF A LARGE KINDRED WITH PHYSIOLOGICAL OBSERVATIONS AND A REVIEW OF THE LITERATURE. (Eng.)

Goldsmith, R. E. (Univ. Cincinnati Coll. Medicine, Room 5564, 231 Bethesda Ave., Cincinnati, OH 45267); Sizemore, G. W.; Chen, I.-W.; Zalme, E.; Altemeier, W. A. *Ann Intern Med* 84(1): 36-43; 1976.

A study was conducted on 69 members of a kindred of 100 members (covering five generations) in which 34 members were hypercalcemic. Sixteen subjects were believed, on the basis of laboratory and clinical observations, to have primary hyperparathyroidism. Eight patients were subjected to exhaustive study to identify polyendocrine involvement before neck exploration. No coexisting endocrine abnormalities were found; operation showed multiparathyroid gland involvement in most instances. Measurement of immunoassayable calcitonin and assessment of renal and gut function were carried out in 37 subjects to search out possible causes of ''reactive'' parathyroid gland hyperfunction. While no such cause-effect relationship was noted for this kindred, 16 subjects so tested had serum calcitonin content below the assay limits of sensitivity. The data are too limited to conclude either that calcitonin synthesis (or secretion) was suppressed or was congenitally absent in these patients.

4624 RETROPERITONEAL GANGLIONEUROBLASTOMA: A KALEIDOSCOPE OF NEURONAL DEGENERATION--A LIGHT AND ELECTRON MICROSCOPIC STUDY. (Eng.)

Powers, J. M. (Medical Univ. South Carolina, 80 Barre St., Charleston, SC 29401); Balentine, J. D.; Wisniewski, H. M.; Terry, R. D. *J Neuropathol Exp Neurol* 35(1): 14-25; 1976.

The light and electron microscopic features of a unique retroperitoneal ganglioneuroblastoma in a 4-yr-old girl are described. The unprecedented concurrence of Hirano, zebra, membranous cytoplasmic (MCB), and Pick bodies in the same population of neoplastic, sympathetic ganglion cells provided evidence for their nonspecificity. Although the pathogenesis of the membranous cytoplasmic bodies in this tumor is unclear, they ostensibly arise within endoplasmic cisterns, similar to the proposed origin of membranous cytoplasmic bodies in Tay-Sachs disease. Both the apparent continuum between argyrophilic bodies and central chromatolysis, and the incorporation of various cytoplasmic constituents within neurofilamentous proliferations reflected some of the dynamic factors involved in the formation of Pick bodies.

4625 ELECTRON MICROSCOPY OF A HEPATIC GANGLIONEUROBLASTOMA WITH SPECIAL REFERENCE TO LONG SPACING COLLAGEN AND TO PORTAL HYPERTENSION. (Eng.)

Knapp, W. A. (Dept. Pathol., Univ. California Medical Center, Sacramento, CA); Ruebner, B. H. *Beitr Path Bd* 157(2): 200-207; 1976.

The case history (including liver biopsy analyses) is presented of a patient with a ganglioneuroblastoma containing neoplastic cells that histologically and ultrastructurally retained Schwann cell characteristics. The Schwann cells produced an unusual type of collagen with a long-spaced cross-banding periodicity of 1,100-1,300 A. This collagen was present throughout the liver and was often directly contiguous with the basement membrane surrounding the stromal tumor cells. In addition to the long-spaced collagen, the interstitial space also contained regular collagen and microfibrils. The regular collagen stained much more strongly with PTA (phosphotungstic acid) than did the long-spaced collagen. The longer periodicity may have resulted from changes in the ground substance chondroitin sulfate concentration. This unusual collagen has been reported in association with other CNS and peripheral nervous system neoplasms and may be useful in the diagnosis of unusual tumors. (10 references)

4626 HEMANGIOBLASTOMA OF THE SPINAL CORD: REVIEW AND REPORT OF FIVE CASES. (Eng.)

Browne, T. R. (Massachusetts General Hosp., Boston, MA 02114); Adams, R. D.; Roberson, G. H. *Arch Neurol* 33(6): 435-441; 1976.

The case histories of five hemangioblastomas of the spinal cord (three women and two boys, 47, 35, 17, 14, and 14 yr old, respectively) are presented, along with a summary of the data from eighty cases in the literature. Median age at onset of symptoms was 30 yr. The ratio of men to women was 1.1:1. Clinical symptoms were usually radicular pain or posterior column sensory loss or both. The lesions were most often single (79%) or intramedullary (60%) and were located in the cervical or thoracic spinal cord. There was associated syringomyelia in 67% of intramedullary cases and meningeal varicosities in 48% of all cases. Lindau disease and hemangioblastomas in other CNS locations were present in 33%. The characteristic roentgenographic picture is a densely vascular tumor in association with a large avascular syrinx and meningeal varicosities. Surgery is the only definitive treatment.

4627 PERIPHERAL FINE STRUCTURE OF A CEREBELLAR HEMANGIOBLASTOMA. (Fre.)

Lefranc, G. (U.E.R. de Medicine et Techniques Medicales, Universite de Nantes, F 44035 Nantes Cedex, France); Pradal, G.; Resche, F.; Mathe, J. F. *Neurochirurgie* 21(5): 365-375; 1975.

he peripheral fine structure of a cerebellar heman-
ioblastoma (CHB) in a woman born in 1947 was
tudied with an electron microscope. During the
eriod 1968-1970 she was hospitalized twice for
ntracranial hypertension syndrome (IHS); vertebro-
asilar angiography showed left localization of a
HB, which was removed surgically each time. A third
peration in 1971 for IHS associated with CHB was
ollowed by fatally progressive cachexia. Peripheral
issue samples were fixed in 3% osmic acid and
tained with uranyl acetate and lead citrate. The
issues showed variably altered cellular bodies,
ost without nuclear membranes, and intricate glial
rojections. Dense capillary basal membranes were
ccompanied by pinocytotic vesicles and numerous
ytoplasmic fibers with involution. There was not
nough morphological modification to provide informa-
ion on CHB histogenesis.

628 THE HUMAN MENINGIOMA: A BENIGN TUMOR WITH SPECIFIC CHROMOSOME CHARACTERISTICS. (Eng.)
ark, J. *In: Chromosomes and Cancer*. Edited by J.
erman. (New York; John Wiley & Sons): pp. 497-517;
974.

ecent cytogenetic findings from 23 meningiomas are
eported and surveyed in relation to 82 cases report-
d earlier. It appears that meningiomas have a tend-
ncy to develop hypodiploid stemlines. The initial
tep is the loss of a No. 22 chromosome. Subsequent
teps affect chromosomes of groups C and D and some-
imes Nos. 1 and 17 or 18. Structural aberrations
re frequent, often affecting group G chromosomes.
ne formation of dicentrics is common, especially as
result of translocation of group F or G chromo-
omes onto group A or C chromosomes. The importance
f dicentrics in the origin of the ring chromosomes
ound in several stemlines and the significance of
arious new marker chromosomes frequently found in
eningiomas are discussed. (21 references)

29 FOLLOW-UP STUDY OF PATIENTS TREATED BY X-RAY EPILATION FOR TINEA CAPITIS: RESURVEY
POST-TREATMENT ILLNESS AND MORTALITY EXPERIENCE.
ng.) Shore, R. E. (New York Univ. Medical Center,
0 First Ave., New York, NY 10016); Albert*, R. E.;
asternack, B. S. *Arch Environ Health* 31(1): 21-28;
976.

second follow-up study of 2,215 persons who during
ildhood had been given x-ray therapy for tinea
pitis and of 1,395 persons matched for age, sex,
d race who were treated for the same disease with-
t the use of x-ray therapy is presented. All per-
ns were treated between 1940 and 1959. An excess
cidence in the irradiated cases of tumors of the
ad and neck was found. There were eight neurogenic
mors, four parotid tumors, and six thyroid adenomas
ported in the irradiated group, whereas no tumors
these areas were reported among controls. There
s a total of 13 skin tumors in irradiated individu-
s and three in controls. The death rate from cancer
the two groups was very similar at 0.32% in the
radiated and 0.29% in the control. Thus, the radia-
on-induced excess of tumors has so far been relative-
nonlethal. Among white patients, a 40% excess of
treated psychiatric disorders was observed in the irradiated group, but no differences occurred among blacks.

4630 CARCINOMA SPINOCELLULARE DYSKERATOTICUM SEGREGANS CUTIS. (Pol.) Jaskiewicz, K. (Instytut Patologii AM, 80-335 Gdansk-Oliwa, ul. Sztormowe 7a/23, Poland); Zgierski, J. *Przegl Dermatol* 62(6): 765-771; 1975.

Over a 6-yr period, eight cases of carcinoma spinocellulare dyskeratoticum segregans cutis, a rare variety of squamous cell cancer, and two cases of dyskeratoma, were confirmed by biopsy material obtained mainly from men aged 45-85 yr. Neoplasms were located most frequently on the facial skin, with diameters of the tumor centers ranging from 3 to 22 mm in seven cases and reaching 9 cm in one case. Histologic analysis of all cancers and dyskeratoma showed excessive cornification of the epidermis and collagen substrate degeneration, disorganized growth of the epidermis, acantholysis, dyskeratosis, and tumorous growth. The cancers were characterized additionally by foci composed of basophilic cylindrical cells or secondary cylindrical cells, with a predominance of structures imitative of hair follicles or new sebaceous gland structures. The findings suggest an adnexal origin of the neoplasm. (8 references)

4631 ANGIOSARCOMA OF THE SKIN: A CLINICOPATHOLOGIC AND FINE STRUCTURAL STUDY. (Eng.) Rosai, J. (Univ. Minnesota Medical Sch., Box 609, Mayo Memorial Building, Minneapolis, MN 55455); Sumner, H. W.; Kostianovsky, M.; Perez-Mesa, C. *Hum Pathol* 7(1): 83-109; 1976.

Ten cutaneous angiosarcomas were presented. These tumors involved the scalp or face of elderly individuals, where they presented as bluish or violaceous plaques and nodules. They had a marked tendency for local spread in surface and depth, a third of them eventually gave rise to distant metastases (particularly to cervical lymph nodes and lung). Microscopically, angiomatous areas of freely anastomosing channels lined by atypical endothelial cells were seen alternating with Kaposi-like spindle cell areas and undifferentiated foci. By electron microscopy, the tumor cells were seen to have all the features of endothelial cells (including pinocytotic vesicles, tubulated bodies, and in one case closed fenestrations). They also exhibited a cytoplasmic specialization, interpreted as the intracellular formation of a vascular lumen. Pericytes and cells resembling smooth muscle cells were also present. In the differential diagnosis, this entity was distinguished from other clinical types of angiosarcoma of the skin and from a number of benign and malignant conditions. Surgery should be used for solitary, well circumscribed tumors and radiation therapy for tumors either multicentric or with ill defined margins.

4632 ANALYSIS OF 24 CASES OF XERODERMA PIGMENTOSUM OBSERVED IN THE BIZERTE REGION. (Fre.) Stephanov, M. S. (Tunis, Tunisia). *Bull Soc Fr Dermatol Syphiligr* 82(5): 414-416; 1975.

Xeroderma pigmentosum, a congenital skin disease seemingly due to a recessive gene, has long been documented in medical literature, but its biological process remains unclear. Some tried to connect it with syphilis, others with adrenal insufficiency or with membership in blood group O. The skin of patients with this disease appears to be highly sensitive to UV rays. The disease frequently evolves into basocellular or spinocellular epitheliomas with metastases. This article describes 24 cases drawn from 8 families living near Bizerte, Tunisia. Ages ranged from 18 mo to 26 yr in 11 men and 16 women. In 78% of the patients, hereditary transmission could be proved. Biopsies revealed the existence of epitheliomas in 5/24 persons. The determination of blood group in 21 cases showed that 14 belonged to group A, 6 to group O, and 1 to group B. Electrophoresis disclosed no hemoglobin pathology. No evidence of syphilis was found.

4633 THE PROBLEM OF GENESIS OF ORBITAL MELANOCYTOBLASTOMA. (Pol.) Tost, M. (Ophthalmology Clinic, Martin Luther Univ., Halle/Saale, East Germany). *Klin Oczna* 45(9): 1003-1009; 1975.

The observation of a 59-yr-old woman with orbital melanocytoblastoma, as well as a review of the literature and observations with experimental animals, suggests that primary melanocytoblastomas in the orbit should be differentiated from secondary ones arising from the skin, conjunctiva, cornea, and choroid. Anatomopathologic and developmental research on animals indicates that primary melanocytoblastomas in the orbit are possible, but they are generally not detectable in the early stage in man. Primary orbital melanocytoblastoma in the case reported was confirmed on the basis of a picture of the eye with adjoining orbital neoplastic tissue. Animal investigations show that dysgenetic extension of cells comprising melanin in extrabulbar orbital tissue is possible; experiments on ABJ mice may serve as a model. A correct diagnosis is possible only through histologic study following orbitotomy, with priority given to rapid absorption in paraffin by freezing. (22 references)

4634 SMALLPOX VACCINATION AND MALIGNANT SKIN TUMORS: A CASE OF MALIGNANT MELANOMA ON A JENNERIAN REVACCINATION SCAR. (Fre.) Labouche, F. (Hopital d'Instruction des Armees de Brest, Brest, France); Chastel, C.; Missoum, A.; Normand, P. *Bull Soc Fr Dermatol Syphiligr* 82(5): 464-468; 1975.

Various malignant skin cancers (baso- or spinocellular epitheliomas, melanomas, or sarcomas) may appear on the scars of smallpox vaccinations or revaccinations. A tumor is described that formed over a scar on the left arm of a 28-yr-old man 5 yr after revaccination and evolved slowly during three additional yr. After a sudden, rapid increase, the growth became painful and the patient sought treatment. A firm black tumor, 6 mm in diameter, was removed and identified as a malignant melanoma. Fifteen months later, there was no local recurrence or adenopathy. Tumors over a jennerian vaccination area seem to occur predominantly in Caucasian men. The records indicate a wide age range, from 15 to 81, with about half of the patients between 40 and 60. The top of the arm remains virtually the only location. The delays, from 20 to 50 yr after primary vaccination, shorten considerably after a revaccination (3 wk to 8 yr). There are known examples of tumors immediately following vaccination. Various hypotheses exist as to the pathogeny of these small neoformations. The vaccination itself, assimilated to a single trauma, may result in a tumor. Some hint at a possible oncological role of the vaccinal virus. Others involve the scar or give pure chance as an explanation. Despite the minuscule number of such tumors compared to the total number of vaccinations, mere coincidence was not accepted as an explanation. It is advocated that smallpox vaccination be done only on healthy skin (never on the heel, a site favored by malignant melanoma) and that surveillance of the scar be maintained in cases of revaccination late in life.

4635 BENIGN CEMENTOBLASTOMA. (Eng.) Corio, R. L. (Natl. Naval Dental Center, Bethesda, MD 20014); Crawford, B. E.; Schaberg, S. J. *Oral Surg* 41(4): 524-530; 1976.

The occurrence, clinical presentations, and treatment of benign cementoblastoma are discussed. The case of a 19-yr-old Caucasian male is noted for the unusual location of the tumor, which completely embedded the impacted maxillary third molar and totally replaced the soft tissue of the pulp. Normally the growth presents as a well-circumscribed radiopaque mass attached to the root(s) of an erupted permanent tooth. These growths occur more frequently in males, with the molar-premolar region of the mandible being most affected. Pain, expansion and swelling of the jaw, and facial asymmetry are the most common clinical features. Although the lesion has unlimited growth potential, a conservative surgical procedure with extraction of involved teeth is suggested because the tumor is easily enucleated from the adjacent bone. According to the data reviewed, this lesion may be more common than previously thought.

4636 SUPRAGLOTTIC LARYNX AND ITS PATHOLOGY AS STUDIED BY WHOLE LARYNGEAL SECTIONS. (Eng.) McDonald, T. J. (Mayo Clinic, Rochester, MN 55901); De Santo, L. W.; Weiland, L. H. *Laryngoscope* 86(5): 635-648; 1976.

To determine the modes of extension of supraglottic carcinomas, whole-organ serial sections were made of 40 human larynges containing this disease whose involvement met clinically accepted criteria for supraglottic laryngectomy. Three case histories (one woman and two men aged 46, 57, and 57 yr, respectively) are also presented. Most of the supraglottic cancers (29) were typically well-differentiated tumors that remained confined to the supraglottic larynx. Exceptions (11) were exemplified by tumors manifesting submucosal extension some distance away from the main tumor mass, tumors invading the thyroid cartilage, second primaries, and tumors disseminating emboli away from the main tumor. Present preoperative diagnostic measures are unable to detect tumors with

:ypical behavior. Subsequent supraglottic laryngectomy
ı patients with such tumors would, therefore, leave
esidual tumor. Carcinomas exhibiting atypical behav-
or were characteristically undifferentiated and
ggressive. These cancers easily invade the epiglottis
ıd preepiglottic space: the latter is removed during
ther supraglottic or total laryngectomy, the recom-
ended treatment. The thyroid cartilage is an excel-
ent barrier to the spread of supraglottic cancers.
ımors that invade it penetrate the anterior commis-
ıre first. The problem in selecting candidates for
ıpraglottic laryngectomy is one of assessment; the
ımor mass makes it difficult to see its full mucosal
ktent. Inadequate biopsy may also fail to detect a
ımor. In the preoperative assessment, supraglottic
ıryngectomy is contraindicated if the biopsy shows
gh-grade differentiation and if the tumor is situa-
ed near the petiole. Undetected extension submucosally
the level of the glottis can result in some failures
th conservation surgery of the larynx.

537 GIANT CELL CARCINOMA OF THE LUNG: A LIGHT AND ELECTRON MICROSCOPIC STUDY. (Eng.)
ıng, N.-S. (Dept. Pathology, McGill Univ., 3775
ıiversity St., Montreal H3A 2B4, Quebec, Canada);
emayer, T. A.; Ahmed, M. N.; Knaack, J. *Hum Pathol*
1): 3-16; 1976.

ıree cases of giant cell carcinoma of the lung and
x other lung carcinomas with large atypical cell
ormation were studied by both light and electron
croscopy. Giant cell carcinomas showed unique light
ıd electron microscopic features; large atypical
lls from other lung carcinomas usually retained some
trastructural characteristics of their cellular
igin. The giant cell carcinoma was characterized
abundant mitochondria, a concentric whorl of
ınofilament-like fibrils, and aggregates of several
irs of centrioles. The ''phagocytic activity'' of
e giant cell carcinoma was attributable to the
-called tumor cell-tumor cell or WBC-tumor cell
ıperipolesis. Giant cell carcinoma of lung could
iginate from a primitive multipotential cell in
e distal bronchiole or be associated with a squa-
us, glandular, or clear cell pattern. Whether in
ıre or mixed form, this tumor should still be con-
dered a specific entity due to its unique light
d electron microscopic features and its fulminant
inical course.

538 ON THE ORIGIN OF THE SO-CALLED TUMORLETS OF THE LUNG. (Eng.) Bonikos, D. S. (Stan-
rd Univ., Stanford, CA); Archibald, R.; Bensch, K.
Hum Pathol 7(4): 461-469; 1976.

light and electron microscopic study of multiple
ımorlets in a 66-yr-old woman is presented. Clin-
ally, the patient had had chronic obstructive lung
sease with bronchiectasis of the right upper lobe.
ide from severe bronchiectatic alterations, the
sected lobe showed typical tumorlets. On electron
croscopic study, these were shown to consist of
lls containing large numbers of a neurosecretory
pe of granule, which was identical to those present
the bronchial counterpart of the intestinal argentaffin (Kulchitsky) cell, as well as in pulmonary carcinoid and oat cell carcinoma cells. The possibility of a histogenic relationship among tumorlets, bronchial carcinoid tumors, and oat cell carcinoma is suggested, the cell of origin in each case being the bronchial counterpart of the intestinal argentaffin cell. (54 references)

4639 INCIDENCE AND TRAITS OF CHRONIC GASTRITIS IN RELATIVES OF PATIENTS WITH GASTRIC CARCINOMA. (Rus.) Salupere, V. P. (Tartu Univ., Tartu, USSR); Kataeva, V. M. *Ter Arkh* 48(3): 86-88; 1976.

The incidence and characteristics of chronic gastritis were investigated in 42 relatives of 16 patients with gastric carcinoma (78% of all living first-degree relatives) and in 46 patients with chronic gastritis whose relatives also suffered from gastric carcinoma. Spot biopsy of the antral and fundal mucosa was performed. The control group contained the following: 66 patients with chronic gastritis whose relatives did not suffer from gastric carcinoma, and 47 persons selected from the medical population and matched for age and sex with the relatives of patients with gastric carcinoma. Fluorescent antibodies to parietal cells of the stomach were found in only two relatives of patients with gastric carcinoma, although 97% of the relatives suffered from chronic gastritis. Gastritis was established in 31/47 control subjects from the medical population. However, the two groups differed significantly only in the incidence of antral gastritis, which appeared more often in the relatives of patients with gastric carcinoma. Not a single case of isolated fundal gastritis was observed among patients with chronic gastritis whose relatives suffered from gastric carcinoma, while it was seen in 11 patients without familial cancer history. The incidence of isolated antral and diffuse gastritis was not dependent upon familial cancer history. (17 references)

4640 CANCER OF THE GASTRIC STUMP. (Spa.) De Arzua Zulaica, E. (Bilbao, Spain). *Rev Esp Enferm Apar Dig* 47(1): 47-84; 1976.

Previous clinical knowledge of cancer of the gastric stump is highlighted and details from 17 actual cases are given. The age of the patients was 40-80 yr, median age 58.8 yr, and only two were females. Median time between original surgery and evidence of cancer was 19 yr. Only seven patients reported epigastic pain and four reported an epigastric disturbance. Other symptoms included thinning of the stump, vomiting, melenas, hematemesis, burning, acidity, and progressive dysphagia. The symptoms began 17 days to 1-1/2 yr before diagnosis. Radiological diagnosis was made in 14 cases; the remaining 3 were made by laparotomy. In four cases, gastroscopic examination confirmed the diagnoses. Of the laparotomies performed, five were inoperable, two had derivation, two had a subtotal resection, and two had a total resection. The remaining five cases were considered inoperable and the patients died within 4 mo. One of the 17 patients has survived the 22-mo postoperative period. It is suggested that in gastric stump patients with gastrointestinal symptoms, a better coordination of radiology, endoscopy, biopsy, and cytology will enable better diagnoses and results after surgery. (125 references)

4641 FECAL STEROIDS AND BACTERIAL FLORA IN PATIENTS WITH POLYPOSIS COLI. (Eng.) Watne, A. L. (West Virginia Univ. Medical Center, Morgantown, WV 26506); Lai, H.-Y. L.; Mance, T.; Core, S. *Am J Surg* 131(1): 42-46; 1976.

Fecal neutral and acid steroids and bacterial flora were studied in 15 patients with polyposis coli and in 14 normal controls. Purification and quantification of bile acids and neutral steroids were carried out by gas-liquid chromatography. The patients with polyposis coli showed a higher concentration of cholesterol and primary bile acids, which increased still more after ileorectostomy. The bacterial flora of the patients with polyposis coli showed an anaerobe/aerobe ratio of 2.7/1 with a relative increase in clostridia and bifidobacteria and decrease in eubacteria and bacteroides. After ileorectostomy, clostridia disappeared as did rumenococcus, peptostreptococcus, and fusobacteria; eubacteria and lactobacilli decreased and bifidobacteria and bacteroides increased after ileorectostomy. Eubacteria showed a decrease from normal in the pre- and postoperative patients with polyposis coli and paralleled the reduction of cholesterol to coprostanol.

4642 HYPERNEPHROMA IN A 6-YR-OLD CHILD. (Spa.) Ochoa, E. (Hospital Roberto del Rio, Area Norte, U. de Chile, Chile); Arribas, R.; Neura, M.; Mirkin, D. *Rev Chil Pediatr* 45(2): 123-126; 1974.

Most monosymptomatic hematurias are difficult to diagnose; the case of a 6-yr-old boy with renal cell carcinoma (rare in pediatrics) is presented. Sporadic episodes of hematuria and throbbing of the left kidney occurred before a pyelogram revealed a semilunar defect in the left renal pelvis 18 mo later. A selective angiography led to the diagnosis of a malignant renal neoplasm. The hyperfusion in the parenchyma surrounding the tumor was probably due to blood flowing from the healthy renal tissue to the carcinoma. Changes in the vascularization provoked by the tumor caused alteration in the kidney. A 90 g kidney and four lymphatic lymph nodes were removed; they confirmed the diagnosis of renal cell carcinoma with invasion of the lymph nodes. There were no further complications as of 8 mo after surgery; however, in some cases metastasis occurs several years later. (17 references)

4643 AN AFLATOXIN IN THE LIVER OF A PATIENT WITH REYE-JOHNSON SYNDROME. (Eng.) Chaves-Carballo, E. (Univ. Iowa Coll. Medicine, Iowa City, IA); Ellefson, R. D.; Gomez, M. R. *Mayo Clin Proc* 51(1): 48-50; 1976.

The aflatoxin contents of liver specimens from eight patients with Reye-Johnson syndrome, two patients with acute encephalopathy, and 10 patients without liver disease were analyzed. Aflatoxin was isolated from the liver of one patient, a 15-yr-old girl who had clinical, laboratory, and pathologic features of Reye-Johnson syndrome. Thin-layer chromatography, fluorescence emission, infrared spectroscopy, and derivatization studies characterized the aflatoxin as related structurally to, but not identical with, aflatoxin B_1. This report is added to previous ones from Thailand, New Zealand, and Czechoslovakia in which the association of aflatoxins and fatty liver has been found in Reye-Johnson syndrome.

4644 PRIMARY LIVER CARCINOMA IN A 16-YEAR-OLD BOY. (Pol.) Tabaszewski, A. M. (Instytut Patologii, Sl. Ak. Med., ul. 3 Maja 13/15, 41-801 Zabrze, Poland). *Wiad Lek* 29(11): 1029-1031; 1976.

Primary liver carcinoma in a 16-yr-old boy was complicated by disturbances of the carbohydrate metabolism. The disease had its onset 3 mo following infectious hepatitis, with the ocurrence of sudden loss of consciousness, convulsions, and enuresis. Inpatient iv administration of a 40% glucose solution relieved the symptoms only temporarily. Hypoglycemia (24-30 mg%) was detected during loss of consciousness, and tumors and ascites characterized the subsequent course. Liver biopsy revealed a malignant tumor. The patient died 7 wk after admission. A section performed 12 hr after death showed a brittle tumor, approximately 16 cm in diameter, connected to the wall of the gallbladder. Microscopic examination indicated predominant solid foci of atypical neoplastic cells similar to liver cells. Anatomopathologic investigation confirmed primary liver carcinoma with metastases to regional lymph nodes and the left lung. (No references)

4645 DOES BRANCHIOGENIC CARCINOMA REALLY EXIST?. (Eng.) Compagno, J. (Armed Forces Inst. Pathology, Washington, DC 20306); Hyams, V. J.; Safavian, M. *Arch Pathol Lab Med* 100(6): 311-314; 1976.

The clinical, gross, and microscopical features of 22 cases (5 women and 17 men; mean age 56 yr, range 40-77 yr), of cystic squamous cell carcinoma of the neck that originally were considered branchiogenic are reviewed. Detailed follow-up investigation demonstrated subsequent distal primary sources of these cervical carcinomas in 19 patients. The primary tumors were located mainly in the oronasopharynx (Waldeyer ring), although primary neoplasms were also found in the lungs, larynx, thyroid, and a major salivary gland. These results support the hypothesis that branchial cleft carcinoma is extremely rare and difficult to substantiate. They also indicate that the cystic metastatic growths that are poorly differentiated or are nonkeratinizing squamous cell carcinomas arise primarily from the oronasopharynx, while those made up of more differentiating tumors may arise from the endolarynx and hypopharynx, thyroid, major salivary glands or the lungs. The long time between metastasis and recognition of the primary lesion indicates that close followups should be performed. The diagnosis of branchiogenic carcinoma should be avoided for cystic squamous cell carcinomas discovered in the neck; they should also be considered metastatic. Empiric radiation of the Waldeyer ring is the recommended treatment to accompany treatment of the neck lesion.

4646 ISOANTIGEN LOSS IN CERVICAL NEOPLASIA: DEMONSTRATION BY IMMUNOFLUORESCENCE AND IMMUNOPEROXIDASE TECHNIQUES.

(Eng.) Bonfiglio, T. A. (Univ. Rochester Medical Center, Rochester, NY 14642); Reinberg, M. R. *Arch Pathol Lab Med* 100(6): 307-310; 1976.

The feasibility of immunofluorescence (IF) and immunoperoxidase (IP) techniques was evaluated in visualizing isoantigen loss during the development of cervical neoplasia. Seventy tissue sections from normal uterine cervices (19 cases) and from uterine cervices with dysplasia (20), carcinoma in situ (19), and invasive cancer (12) were studied for the presence of isoantigens A and B. Isoantigens were demonstrated in normal squamous epithelium. Antigens were lost in foci of invasive cancer, considerably reduced or lost in areas of classic carcinoma in situ in all cases, and reduced in most cases of dysplasia. These observations confirm those obtained with the specific red cell adherence test. The results suggest that the IF and IP techniques are sensitive, reproducible, easily performed methods for demonstrating the loss of isoantigens in cervical neoplasia. The IP method may be more advantageous than the IF method in that the preparations are permanent and can be easily stored.

4647 THE APPLICATION OF ORGAN CULTURE TO THE STUDY OF CELLULAR EVENTS IN THE UTERINE CERVIX.

(Eng.) Fink, C. J.; Thomas, G. H. *In: Gynaecological Malignancy: Clinical and Experimental Studies*. Edited by M. G. Brush; R. W. Taylor. (Baltimore: Williams and Wilkins): pp. 63-86; 1975.

The behavior of human cervical explants in organ culture has been investigated by standard histological methods, scanning electron microscopy, and autoradiography in order to develop a model system for studying metaplasia and dysplasia of the cervix. Normal squamous epithelium from the ectocervical area remained well-preserved for up to 6 days in culture. However, a progressive loss of superficial and intermediate cells was apparent even in the presence of estradiol. This progressive desquamation reflects a failure of the in vitro system to mimic the in vivo situation, in which a thickened epithelium is maintained, particularly under estrogen stimulation. The premitotic incorporation of thymidine by basal cells continued for at least 30 hr in culture. Explants of endocervical columnar epithelium survived well in culture for up to 8 days, although some loss of cell height was evident. On the more exposed portions of the endocervical explants, stromal and columnar changes become very marked; the changes were suggestive of in vivo metaplasia after 2 to 3 days, with many explants demonstrating an apparent breakdown of the distinction between columnar and underlying cells. Organ culture may thus provide an important system with which to investigate the origin of cells forming metaplastic epithelium and the morphogenesis of cervical dysplasia, which has been postulated to be a stage of cervical cancer. (51 references)

4648 SOME ULTRASTRUCTURAL ASPECTS OF CLEAR-CELL UTERINE CORPUS CARCINOMA.

(Eng.) Liebhart, M. (Medical Acad. Warsaw, Warsaw, Poland); Szamborski, J. *Pol Med Sci Hist Bull* 15/3(4): 399-405; 1975.

A clear-cell carcinoma of the corpus uteri excised from a 71-yr-old woman was studied by light and electron microscopy to compare the clear cell areas with the fields of less mature, nonsecretory darker cells. Under light microscopy, the clear cells of the tumor showed a high degree of cellular differentiation, with large, empty cytoplasms arranged in solid areas or glandular formations. Ultrastructurally, the nuclei were oval, with chromatin arranged as in normal cells. The cytoplasm contained numerous ribosomes without contact with the endoplasmic reticulum. In the dark cell areas of the tumor, the tissue was composed of smaller columnar or more pleomorphic cells arranged in papillary or irregular glandular formations. The cytoplasm was scanty and eosinophilic; the nuclei differed in shape and were characterized by irregularly dispersed chromatin. Generally, the lysosomes, mitochondria, and ribosomes were more numerous compared to the clear cells. The tissue in this area showed only a slight tendency to glandular arrangement. No accumulation of lipids and lysosomal bodies, characteristic of adrenal gland cells, was observed in the cytoplasm of the carcinomatous clear cells, distinguishing them from mesonephric derivatives. It is concluded that the clear cells of corporeal adenocarcinoma resemble secretory endometrial glandular cells and that they show a high degree of cellular differentiation compared to typical carcinomatous dark cells. The clear cells of the tumor can be distinguished from the mesonephric epithelium and from adrenal gland cells by electron microscopy.

4649 IN VITRO STUDIES ON ENDOMETRIUM, ENDOMETRIAL AND OVARIAN CARCINOMA.

(Eng.) Crocker, S. G.; Milton, P. J. D.; Taylor, R. W.; King, R. J. B. *In: Gynaecological Malignancy: Clinical and Experimental Studies*. Edited by M. G. Brush; R. W. Taylor. (Baltimore: Williams and Wilkins): pp. 179-193; 1975.

The content of nuclear and cytoplasmic progesterone and estradiol receptors has been investigated in normal human endometrial tissue throughout the menstrual cycle and in endometrial cystic hyperplasia and endometrial carcinoma tissues. Estradiol receptors were observed in the cytosol and nucleus of normal tissue; they were present in large amounts during the follicular phase of the ovarian cycle (with a maximum near ovulation), followed by a decline in the luteal phase. Estradiol binding levels were approximately 0.5 unit for days 1-10 of the cycle, 1.8 units for days 11-14, 1.2 units for days 15-21, and 0.8 unit for days 22-28. Cystic hyperplasia tissue gave levels in the lower range of normal values (mean 0.7 unit for 12 cases), irrespective of the duration of amenorrhea. The values for carcinoma were also in the normal range (mean 1.3 units for 11 cases), irrespective of the state of tumor differentiation. All cases of primary and secondary ovarian carcinomas, two cases of cervical carcinoma, and one ovarian fibroma showed no significant binding. One adenoacanthoma of the endometrium showed binding in the low normal range. Assays of nuclear receptor levels demonstrated the same trends as those for cytoplasmic receptors, suggesting that the nuclear uptake of estradiol depends on the availability of cytoplasmic receptors. A specific 5S-8S receptor complex for progesterone was found in large amounts (4-13 units) in normal endometrium during the follicular phase, with a fall in the luteal phase (to 0.3-7 units). In cases of cystic hyperplasia the mean receptor content was comparable with that of normal tissues in the follicular phase, reflecting either prolonged but mild estrogen stimulation or the absence of endogenous progesterone capable of blocking available sites. Endometrial carcinoma tissues from both pre- or postmenopausal patients contained progesterone receptors, with the exception of one anaplastic tumor. The receptor content seemed to be related to the state of tumor differentiation and may reflect tumor responsiveness to progestogen therapy. (29 references)

4650 ENDOMETRIAL CYSTIC HYPERPLASIA--A PREMALIGNANT STATE OR A COMPLEX ENDOCRINE DISORDER?

(Eng.) Brush, M. G.; Milton, P. J. D.; Swain, M. C.; Moore, J. W. *In: Gynaecological Malignancy: Clinical and Experimental Studies*. Edited by M. G. Brush; R. W. Taylor. (Baltimore: Williams and Wilkins): pp. 155-178; 1975.

The morbid anatomy, histology, and clinical symptoms of endometrial cystic hyperplasia are described, and the possible relationship between endometrial hyperplasia and endometrial carcinoma is discussed. The transition from ''atypical'' hyperplasia to endometrial carcinoma occurs in about]2%-14% of cases, but the transition from ''benign'' hyperplasia to the atypical form is not understood, although both types of hyperplasia have been present in some samples of endometrium. Two main categories of patients with endometrial cystic hyperplasia are seen: (a) premenopausal women with dysfunctional uterine bleeding sometimes accompanied by amenorrhoeic episodes; (b) postmenopausal women with spasmodic episodes of bleeding not explicable by other causes such as cervical or endometrial carcinoma, cervical polypi, and atrophic or infective vaginitis. It is unlikely that premenopausal hyperplasia is associated with consistently raised estrogen levels, because plasma estradiol and estrone levels were essentially normal in 19 cases studies (30-140 pg estradiol/ml, 40-110 pg estrone/ml), although some high values were seen (up to 310 pg estradiol/ml and 431 pg estrone/ml). Plasma estrone levels were significantly raised about 1.5- to 3-fold in two postmenopausal patients. Significantly lower mean levels of total urinary 11-deoxy-17-oxosteroids (2.89; mean 4.31 mg/day), androsterone (1.34; mean 1.84 mg/day), etiocholanolone (1.28; mean 1.69 mg/day), and 17-hydroxycorticosteroids (5.79; mean 8.38 mg/day) were found in premenopausal cystic hyperplasia patients compared to normal controls. No such abnormality in androgen status was seen in the two postmenopausal patients studied. It is possible that lowered androgen levels together with normal or slightly increased estrogen levels could be an etiological factor in premenopausal endometrial hyperplasia. (72 references)

4651 KLINEFELTER SYNDROME AND BREAST CANCER.

(Eng.) Baker, S. B. (Johns Hopkins Hosp., Baltimore, MD 21218); Salyer, D. C.; Klinefelter, H. F.; Cole, E. W. *J Hopkins Med J* 138(3): 102-108; 1976

The case history and treatment regime of a patient with Klinefelter's syndrome and breast cancer are presented. The syndrome was first described in 1941 as being characterized by gynecomastia, small testes, aspermatogenesis, hypoleydigism, and increased follicle-stimulating hormone. The Barr body sex chromatin is present in buccal mucosa smears of patients with Klinefelter's syndrome, and one or more extra X chromosomes are associated with the disease in 80% of the patients. Replacement therapy with testosterone and removal of breast tissue are the treatments of choice. Male breast cancer is extremely rare relative to that in females. The courses of the cancers are similar in both sexes and many of the same factors seem to be operating in both types. Treatments are similar except that male cancers respond more positively to hormonal therapy. There seems to be a positive correlation between Klinefelter's syndrome and breast cancer; males with Klinefelter's syndrome have a much higher risk of breast cancer than normal males.

4652 STRUCTURAL CHANGES IN THE OVARIES IN BREAST CANCER.

(Bul.) Maleva, E. (MA, Med. Fac., Bulgaria). *Onkologia (Sofia)* 12(3): 104-106; 1975.

Structural changes in the ovaries of 28 women, aged 24 to 54, with breast cancer were studied morphologically or histochemically. The patients underwent ovariectomy either during the proliferation or during the secondary phase of the breast cancer. The ovaries of 10 women of reproductive age served as the control group. The established morphological changes were of a single type. In 26 women, hyperplasia of the stromal tissue, diffuse or nodular, with increased lipid content in the interstitial cells, and follicular cysts with hormonal activity were found. The origin of these changes and their relation to the malignant growth in the mammary gland are discussed. A convincing cortical hyperplasia was not found in the control group. The results of this study show that in breast cancer subjects there is hyperplasia of the interstitial cells, both cortical and medullary.

4653 GONADOBLASTOMA WITH TUBAL PREGNANCY.

(Eng.) Pratt-Thomas, H. R. (Medical, Univ. South Carolina, 80 Barre St., Charleston, SC 29401); Cooper J. M. *Am J Clin Pathol* 65(1): 121-125; 1976.

A case of unilateral gonadoblastoma in association ith a ruptured ectopic tubal pregnancy is presented. he patient, a 22-yr-old black woman, had a normal enstrual history prior to a missed period followed y abdominal pain and the passage of clots. The urgical specimen, in addition to the ruptured tubal regnancy, showed nodular foci of typical gonadoblasoma in the right ovary, the remainder of the ovarian issue being normal. The left ovary was subsequently emoved, and examination revealed normal constitunts without evidence of tumor. Cytogenetic evaluaion of peripheral WBC showed cells with a normal emale sex chromatin complement, 46-XX, as well as ells with 45 chromosomes in which one of the X chromoomes was missing. The extent of mitotic activity hown by the germ cells coupled with the lack of ccompanying mesenchymal sex cord proliferation in he small closely grouped germ cell aggregates was oteworthy. These observations may indicate that the otentiality for malignant transformation by this umor was enhanced.

654 THE OCCURRENCE OF MALIGNANCY IN MARRIED COUPLES. (Ser.) Varga, J. (Health Centre 'Novi Sad'', General Med. Service, Novi Sad, Yugolavia). *Med Pregl* 28(7-8): 311-313; 1975.

his paper presents case histories of 10 married ouples who were diagnosed with malignant tumors 1965-1974). The people lived in the same town and nder similar living conditions; all but two couples ere immigrants to the area. Malignancy developed in e spouses within 1-6 yr of each other. Reasons for e phenomenon could not be determined. Some specific ases included: a husband who developed lung cancer 44 and whose wife developed ovarian cancer at 45; husband and wife who both developed gallbladder ancer, at ages 77 and 61, respectively; husband, ng cancer, 56, wife, breast cancer, 49; husband, ancer of the larynx, 64, wife, ovarian cancer, 67; sband, cancer of the pancreas, 74, wife, cancer of e gallbladder, 72; husband, pancreatic cancer, 64, fe, abdominal cancer, 64; husband, cancer of the ostate, 75, wife, breast cancer, 69; husband, ncer of the clavicle, 40, wife, hip cancer, 42; sband and wife, pancreatic cancer, 60 and 50, spectively; husband, hip cancer, 38, wife, breast ncer, 42.

655 EARLY AGE OF ONSET OF FAMILIAL BREAST CANCER: GENETIC AND CANCER CONTROL IMPLICAIONS. (Eng.) Lynch, H. T. (Creighton Univ. Sch. edicine, Omaha, NB); Guirgis, H.; Brodkey, F.; loney, K.; Lynch, P. M.; Rankin, L.; Lynch, J. *Arch rg* 111(2): 126-131; 1976.

spite the fact that breast carcinoma is the most equently occurring cancer in American women and s been the subject of extensive epidemiologic vestigation, little attention has been devoted to e of risk-factor information in its control. Six 52 breast-cancer-prone families had at least one man who had manifested breast cancer at age 30 yr earlier. Once such family had two women with breast ncer before age 30 (and at least 10 others affected age 50). Transmission of the lesion was consistent with autosomal-dominant mode of inheritance. Ages of onset indicated a notably early peak (in contrast with the expected pre- and post-menopausal peaks in findings of the New York State Tumor Registry and others). Genetic and epidemiologic information should be utilized to control breast cancer in certain families.

4656 CHROMOSOME BALANCE AND THE CONTROL OF MALIGNANCY. (Eng.) Bloch-Shtacher, N. (Dept. Genetics, Weizmann Inst. Science, Rehovot, Israel); Sachs, L. *J Cell Physiol* 87(1): 89-100; 1976.

The Giemsa banding pattern of the chromosomes was analyzed in a line of transformed golden hamster cells, revertant and re-revertant cells and their tumors. The transformed and re-revertant cells were malignant in vivo and gained an additional chromosome 5_7. Revertants with a suppression of malignancy lost this additional chromosome 5_7 and gained an additional chromosome 7_2. The tumors produced by segregants from the revertant cells were malignant (although to a lower degree than transformed and re-revertant cells). These tumors lost the additional chromosome 7_2 found in revertants and gained one or two 5_{12} chromosomes. The balance between genes for expression and suppression could control malignancy. Chromosome 7_2 carried genes for suppression, and chromosomes 5_7 and 5_{12} carried genes for expression of malignancy. The genes on chromosome 5_7 to result in a greater degree of expression than the genes on chromosome 5_{12}.

4657 CANCER FAMILY SYNDROME. (Eng.) Lynch, H. T.; Krush, A. J.; Thomas, R. J.; Lynch, J. *In: Cancer Genetics*. Edited by H. T. Lynch. (Springfield, IL: Charles C. Thomas): pp. 355-388; 1976.

Criteria for determining the cancer family syndrome are described. This syndrome is characterized as follows: (a) increased occurrence of adenocarcinoma (primarily of the colon and endometrium); (b) increased frequency of multiple primary malignant neoplasms; (c) earlier than average age at onset of cancer (compared to the general population for the specific neoplasm); and (d) vertical transmission of cancer consistent with an autosomal dominant inheritance pattern. The pedigrees of eight families are presented and discussed. It is recommended that clinicians should search carefully for information concerning familial cancer incidence because the implications for cancer control are rewarding. It is also recommended that investigators report additional families in which the cancer family syndrome is well-documented. (13 references)

4658 THE RELATIONSHIP OF CHROMOSOMAL ABNORMALITIES TO NEOPLASIA. (Eng.) Rowley, J. D. (Div. Biological Sciences, Univ. Chicago, Chicago, IL). *Adv Pathobiol* 4: 67-73; 1976.

The relationship between chromosomal abnormalities and neoplasia is discussed. A consistent chromosomal abnormality has been observed in the marrow cells of patients with much greater variability in acute leukemia and other human tumors. Evidence that the Ph^1 chromosome in CML represents a specific translocation rather than a deletion is presented. Recent evidence from studies of banded chromosomes indicates the presence of an abnormal karyotype in patients with CML. Females lacked an X and, in addition, each patient had a break in No. 8 in the same band. When CML patients enter the acute phase of their disease, one of the most common changes is the addition of one or more C group chromosomes. In six patients examined, each had the same extra chromosome, No. 8; three had two extra C's, the second one being an 8, a 10, or an X (in a male). It is thus possible to distinguish random from nonrandom changes. A frequent association of abnormalities of chromosome No. 8, as an extra chromosome or in a translocation, has been observed with a number of hematologic disorders. This has also been reported to be abnormal in meningiomas and polyps of the colon, although the significance is unclear. Animal studies have indicated that the karyotypic change in rats given dimethylbenzanthracene is agent specific and is independent of the tumor type, since the same abnormal karyotype results regardless of whether the carcinogen is given by a route that produces leukemia or one that produces sarcoma. It is concluded that chromosomal studies may provide important clues regarding the mechanisms of carcinogenesis. (20 references)

4659 ADENOPATHIES IN THE COURSE OF HYDANTOIN THERAPY: HISTOPATHOLOGICAL PROBLEMS. (Fre.)

Lederlin, P. (No affiliation given); Mangin-Cheval, M. D.; Macinot, C.; Thibaut, G. *Ann Med Nancy* 15: 33-34, 37; 1976.

The appearance of adenopathies is one of the undesirable side effects of hydantoin therapy against epilepsy. Deep and superficial ganglia may be involved, but the cervical location seems the most frequent. A diffuse infiltration of reticular, more or less atypical cells supersedes the normal structure of the ganglia. There are disturbing symptoms: persistent fever, asthenia, and loss of weight. It is often difficult to distinguish between an authentic malignant lymphoma and a pseudolymphoma. Replacing hydantoin therapy with another antiepileptic medication usually provides the solution. In the past decade, there have been documented reports of malignant lymphomas following long-term treatment by hydantoins. However, the histopathological diagnosis remains difficult. The case is cited of a 22-yr-old woman hospitalized for adenopathies in the right jugulocarotidian area. The first ganglion biopsy revealed a diffuse homogenization, with numerous lymphocytes, rare polynuclear cells, and plasmocytes. There were also atypical reticular cells, some large with burgeoning or multiple nuclei (Sternberg's cells). The diagnosis was a lymphohistiocytic form of Hodgkin's disease. However, it was learned that the patient was an epileptic, receiving daily doses of Trinuride, which includes 4 cg of phenytoine per tablet. The possibility arose of a pseudolymphoma due to hydantoins. A new medication (carbamazepine) replaced the Trinuride. Two months later, the patient's condition was unchanged. A new ganglion biopsy offered an image of subacute adenitis with hyperplasia of some lymphoid follicles. A third biopsy a month later showed some reticular cells. The other elements of granuloma remained absent. The reluctance to identify the case as Hodgkin's disease continued. The diagnosis remained subacute adenitis with atypical reticulosis.

4660 PROJECTING FROM ANIMAL EXPERIMENTS TO CLINICAL CANCER. (Eng.)

Hewitt, H. B. *In: Fundamental Aspects of Metastasis*. Weiss, L., ed. (Amsterdam: North-Holland Publishing Co.): pp. 343-357; 1976.

In order to extrapolate the findings of animal tumor progression experiments to humans, the animal tumor system must resemble spontaneously occurring human tumors as closely as possible. For this reason, the animal tumor system must not embody forms of resistance that are attributable to laboratory artifacts associated with their induction or conditions of transplantation. Such systems include tumors of unknown genetic origin (e.g., Ehrlich's tumor in mice, Walker 256 tumor in rats, or the V2 carcinoma in rabbits); tumors transplanted outside the inbred substrain of animals in which they arose, which includes most tumors that arose some years previously in some other laboratory; and, finally, tumors induced by powerful chemical carcinogens, most of which exhibit a level of immunogenicity rarely, if ever, displayed by tumors of spontaneous origin transplanted within the isogeneic colony of animals in which they arose. Time scale data are presented for natural metastasis from a strain CBA murine carcinoma surgically removed from the implantation site in the flank after 17-21 days. The pattern of metastasis in the system (local recurrence at the operation site, progressive growth of the tumor in the original lymph node--the axillary node, pulmonary metastases, and systemic metastases) can be extrapolated to clinical observations of tumor progression in man, provided that proper allowances are made for the faster median tumor volume doubling time in mice and the larger body volume of humans. (17 references)

4661 ULTRASTRUCTURAL ANALYSIS OF MALIGNANT CONVERSION IN VITRO OF EPITHELIAL CELLS DERIVED FROM NORMAL ADULT RAT LIVER. (Eng.)

Karasaki, S. (Departement d'Anatomie, Universite de Montreal, Montreal, Canada); Simard, A.; de Lamirande, G. *Eur J Cancer* 12(7): 527-534; 1976.

A tumorigenic epithelial-type cell line was established from a long-term culture of isolated liver cells from a normal adult Wistar rat. The culture medium was Ham's F12 containing 100 U penicillin G and 100 µg streptomycin/ml, supplemented with 5% or 10% fetal calf serum. Electron microscopic examinations of the transformed epithelial cell foci in the primary culture revealed the production of virus particles in association with membranes of the nuclear envelope, endoplasmic reticulum, and cell surface. The epithelial cells were quiescent and they had intranuclear canalicular membranes. By trypsinization, the cells were initiated into a rapid replication and showed subcellular differentiation. In passage 1, annulate lamellae and paired cisternae emerged, and virus production ceased. Close apposition of adjacent cells became prominent in passage 9, when the cell line first revealed tumorigenicity upon transplantation into neonatal Wistar rats. Concomitantly, cytochemical tests gave a positive ATPase reaction on the plasma membranes. This activity continued in the epithelial cells following passages in vitro as well as in tumor cells in vivo. The possible origin of epithelial cells in vitro from hepatocytes was suggested by the demonstration of peroxisomes and bile canalicular-like structures in the carcinoma cells. (46 references)

4662 GENETIC CHARACTERISTICS OF THE HELA CELL. (Eng.) Hsu, S. H. (Johns Hopkins Univ. Sch. Medicine, Baltimore, MD 21205); Schacter, B. Z.; Delaney, N. L.; Miller, T. B.; McKusick, V. A.; Kennett, R. H.; Bodmer, J. G.; Young, D.; Bodmer, W. F. *Science* 191(4225): 392-394; 1976.

4663 INVESTIGATION OF CHROMOSOMAL MUTABILITY IN PATIENTS WITH HIGH RISK OF MALIGNANCY. (Spa.) Schuler, D. (Departamento de genetica, II. Departamento de pediatria, Universidad Medica ''Ignac Semmelweiss'', Tuzolto utca 7-9, Budapest IX, Hungary); Dobos, M.; Fekete, G. *Rev Cubano Pediatr* 47(3): 343-351; 1975.

4664 MALIGNANT BLOOD CELL NEOPLASMS OF GENETIC ORIGIN IN *DROSOPHILA MELANOGASTER* (Meeting Abstract). (Eng.) Gateff, E. (Biologisches Institut I der Universitaet, 78, Freiburg i. Br., Schaenzlestr. 9, West Germany). *J Cell Biol* 70(2/Part 2): 6a; 1976.

4665 RE: A HUMAN SQUAMOUS CELL CARCINOMA CELL LINE (Letter to Editor). (Eng.) Moore, G. E. (Denver General Hosp., Denver, CO 80204); Merrick, S. B.; Woods, L. K.; Arabasz, N. M. *Cancer Res* 36(5): 1849; 1976.

4666 RE: A HUMAN SQUAMOUS CELL CARCINOMA CELL LINE (Letter to Editor). (Eng.) Nelson-Rees, W. A. (Univ. California Sch. Public Health, Oakland, CA 94625). *Cancer Res* 36(5): 1849-1850; 1976.

4667 TROPIC INTERACTIONS BETWEEN HUMAN ENDOTHELIAL AND RAT ASTROCYTOMA CELLS (Meeting Abstract). (Eng.) Savage, K. E. (Dept. Human Biological Chemistry, Genetics, and Cell Biology, Univ. Texas Medical Branch, Galveston, TX 77550). Suddith, R. L.; Baur, P. S.; Hutchison, H. T. *J Cell Biol* 70(2/Part 2): 91a; 1976.

4668 HISTOCHEMICAL AND ULTRASTRUCTURAL INVESTIGATIONS ON ORGAN CULTURE OF MALIGNANT TUMORS. (Ger.) Hecker, D. [Medizinischer Bereich, Humboldt-Universitaet Berlin (Charite), Geschwulstklinik, DDR-104 Berlin, Schumannstrasse 20/21, East Germany]; Deutschmann, A. *Acta Histochem (Jena)* 55(1): 8-13; 1976.

4669 ULTRASTRUCTURAL CHANGES IN CHRONIC LYMPHOCYTIC LEUKEMIA LYMPHOCYTES STIMULATED WITH SODIUM PERIODATE (Meeting Abstract). (Eng.) McGill, M. (American Natl. Red Cross Blood Res. Lab., 9312 Old Georgetown Rd., Bethesda, MD 20014). *J Cell Biol* 70(2/Part 2): 114a; 1976.

4670 THE MITOCHONDRIA AND MITOCHONDRIAL DNA OF HUMAN LEUKEMIC CELLS (Meeting Abstract). (Eng.) Choudhury, R. P. (Biophysics Lab., Saha Inst. Nuclear Physics, Calcutta, 700037 India); Misra, D. N.; Maiti, S.; Sadhukhan, P. *J Cell Biol* 70(2/Part 2): 9a; 1976.

4671 PRELEUKEMIA AND THE LEUKEMIAS AS A MODEL OF CARCINOGENESIS. (Ger.) Schubert, J. C. F. (Zentrum der Inneren Medizin, Johann Wolfgang Goethe-Universitaet, Frankfurt a.M., West Germany); Walther, F. *Therapiewoche* 26(6): 879-885; 1975.

4672 THE HEMATOLOGIC FEATURES OF THE PRELEUKEMIC PHASE OF MYELOMONOCYTIC LEUKEMIA (Meeting Abstract). (Eng.) Pierre, R. V. (Mayo Clinic, Rochester, MN 55901). *Am J Clin Pathol* 65(2): 267; 1976.

4673 PERNICIOUS ANEMIA TERMINATING IN ACUTE MYELOBLASTIC LEUKEMIA. (Eng.) Bergevin, P. R. (Walter Reed General Hosp., Washington, DC 20012); Blom*, J. *South Med J* 69(1): 110-112; 1976.

4674 MANDIBULAR HISTIOCYTOSIS X AND ACUTE LYMPHOBLASTIC LEUKEMIA. (Eng.) Kanter, H. M. (Zoller Dental Clinic, Univ. Chicago, 950 East 59th St., Chicago, IL 60637); Lin, L.-M.; Goepp, R. A.; Olson, R. E. *Oral Surg* 42(2): 221-230; 1976.

4675 AN AUTOPSY CASE OF H.C.G. SECRETING INTRACRANIAL TERATOMA. (Eng.) Hirano, T. (Nagasaki Univ. Sch. Medicine, Nagasaki, Japan); Kawai, K.; Tsuchiyama, H. *Acta Pathol Jpn* 26(1): 97-103; 1976.

4676 GLIOBLASTOMA AND NEUROFIBROMA AFTER EXPOSURE TO NITROGEN MUSTARD. (Ger.) Weiss, A. (D-2000 Hamburg 20, Beim Andreasbrunnen 5, West Germany); Weiss, B. *Munch Med Wochenschr* 118(27): 875-878; 1976.

4677 SYSTEMIC LUPUS ERYTHEMATOSUS AND CARCINOMA OF LUNG. (Eng.) Sumner, P. (Booth Memorial Medical Center, Flushing, NY); Dwek, J. H. *NY State J Med* 76(1): 94-96; 1976.

4678 MORPHOLOGY AND CLINICAL SIGNIFICANCE OF MENINGEAL HEMANGIOPERICYTOMA. (Ger.) Kastendieck, H. (Institut fuer Pathologie, Universitaet Hamburg, Martinistrasse 52, D-2000 Hamburg 20, West Germany); Kloeppel, G.; Altenaehr, E. *Z Krebsforsch* 85(3): 287-297; 1976.

4679 THE POSTERIOR TRIANGLE IN RADICAL NECK SURGERY. (Eng.) Sholnik, E. M. (1855 W. Taylor St., Chicago, IL 60612); Yee, K. F.; Friedman, M.; Golden, T. A. *Arch Otolaryngol* 102(1): 1-4; 1976.

4680 PSEUDOMESOTHELIOMATOUS CARCINOMA OF THE LUNG. (Eng.) Harwood, T. R. (Depts. Pathology, V.A. Res. Hosp., 333 E. Huron St., Chicago, IL 60611); Gracey, D. R.; Yokoo*, H. *Am J Clin Pathol* 65(2): 159-167; 1976.

4681 MULTIFOCAL ADENOID CYSTIC CARCINOMA OF THE LIP. (Eng.) Appel, B. N. (Sch. Dental Medicine, Univ. Pittsburgh, Pittsburgh, PA 15261); El Attar, A. M.; Paladino, T. R.; Verbin, R. S. *Oral Surg* 41(6): 764-771; 1976.

4682 PRIMARY OSTEOMA OF THE SPHENOID SINUS. (Eng.) Mikaelian, D. O. (Jefferson Medical Coll., Philadelphia, PA 19107); Lewis, W. J.; Behringer, W. H. *Laryngoscope* 86(5): 728-733; 1976.

4683 RECENT PROGRESS IN MORPHOLOGY AND DIAGNOSIS OF BONE DISEASES AND BONE TUMORS. (Ger.) Delling, G. (Pathologisches Inst. der Univ., Martinistr. 52, D-2000 Hamburg 20, West Germany); Schulz, A.; Seifert, G. *Radiologe* 16(2): 46-53; 1976.

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4686 ANGIOLEIOMYOMA OF THE SKIN. (Ger.) Bardach, H. (II. Univ.-Hautklinik, Alserstrasse 4, A-1090 Vienna, Austria); Ebner, H. *Hautarzt* 26(12): 638-642; 1975.

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4704 TREATMENT OF EARLY CANCER OF THE BREAST: HISTOLOGICAL STAGING AND ROLE OF RADIOTHERAPY. (Eng.) McDonald, A. M. (King's Coll. Hosp. Medical Sch., Denmark Hill, London SE5 8RX, England); Simpson, J. S.; MacIntyre, J. *Lancet* 1(7969): 1098-1100; 1976.

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4708 CYTOENZYMOLOGY OF BENIGN AND MALIGNANT TUMOURS OF THE CORPUS UTERI. I. RESPIRATORY ENZYMES. (Eng.) El-Fiky, S. M. (Medical Res. Inst., P. O. Box El-Hadra, Alexandria, Egypt); Taha, Y. M. *Acta Histochem (Jena)* 56(1): 1-17; 1976.

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4710 ATYPICAL RESERVE CELL HYPERPLASIA AND THE MORPHOGENESIS OF CARCINOMA OF THE UTERINE CERVIX (Meeting Abstract). (Dut.) Beyer-Boon, M. E. (Leiden, The Netherlands); Verdonk, G. W. *Ned Tijdschr Geneeskd* 119(48): 1917; 1975.

4711 SCANNING ELECTRON MICROSCOPY OF NORMAL UTERINE CERVIX, CARCINOMA IN SITU AND SQUAMOUS CELL CARCINOMA: PRELIMINARY FINDINGS. (Eng.) Foden, A. P. (Dept. Obstetrics and Gynaecology, Univ. of the Witwatersrand, Johannesburg, South Africa); Freedman, R. S.; Abrahams, C. *S Afr Med J* 50(15): 571-573; 1976.

See also:

* (Rev): 4224, 4233, 4240, 4241, 4242, 4243, 4244, 4245, 4246, 4248, 4249, 4250, 4251, 4253, 4254, 4256, 4261, 4263, 4264, 4266, 4267, 4269, 4270, 4282, 4286, 4293
* (Chem): 4321, 4329, 4330, 4340, 4356, 4362, 4368, 4372, 4373, 4376
* (Phys): 4378, 4380, 4381, 4384, 4400, 4403, 4417
* (Viral): 4461, 4480, 4531, 4534, 4549
* (Immun): 4566, 4569, 4586, 4598, 4600
* (Epid-Biom): 4713, 4720, 4721, 4722, 4725, 4726

4712 THE PROBLEM OF CANCER IN INDIA: AN EPIDEMIOLOGICAL ASSESSMENT. (Eng.) Jussawalla, D. J. (Dept. Surgery, Tata Memorial Cancer Centre, Parel, Bombay, India). *Gann Monogr Cancer Res* (18): 265-273; 1976.

Data related to the incidence of various kinds of cancer in different regions of India are presented and compared briefly with the incidences in Connecticut and Japan. In greater Bombay, the overall incidence per 100,000 is 71.8 for men and 67.7 for women and the respective mortality rates per 100,000 are 44.8 and 38.8. The incidence rates in Connecticut are approximately 1.7-1.8 times higher. In Bombay, cancer is the sixth leading cause of death for males and the seventh for females. Among males, oropharyngeal cancer accounts for 49%-73% of all cancers in various districts, while among females genitourinary cancer accounts for 54%-77% (male:female ratio 13:1). Smoking and pan chewing, which have been shown to be associated with oropharyngeal and esophageal cancer, are described briefly. The age-adjusted incidence rates of esophageal cancer for males and females in Bombay are 13.0% and 11.3%, respectively, compared to 5.7% and 1.4% for Connecticut. The age-adjusted incidence rates for stomach cancer for males and females in Bombay are 10.0% and 6.5%, respectively, compared to 14.7% and 6.8% in Connecticut and 95.3% and 44.7% in Japan. The rate of colonic cancer is low, but that of cancer in the rectum and the anal canal is high. It is suggested that elimination of pan chewing and bidi smoking would reduce the incidence of upper alimentary as well as oropharyngeal cancer. Factors associated with an increased incidence of cervical cancer include pregnancy in the middle and late teens and underprivileged social class. The age-adjusted incidence rates of breast cancer in Bombay, Connecticut and Japan are 20.4%, 62.3%, and 11.0%, and the corresponding rates for cervical cancer are 24.7%, 10.3%, and 20.6%. The incidence of breast cancer among Parsi and Sindhi women is higher than in other religions. (3 references)

4713 ORAL AND OROPHARYNGEAL TUMORS. (Eng.) Wahi, P. N. (Indian Council of Medical Res., Ansari Nagar, New Delhi 110016, India). *Gann Monogr Cancer Res* (18): 19-26; 1976.

Factors associated with the incidence of oropharyngeal cancer in India, particularly the use of tobacco and alcohol, are discussed, and the histological types of tumors are outlined. The buccal mucosa is the most common site for these tumors (64.7%). The age-specific incidence for both males and females is higher than anywhere else in the world. The incidence is less in persons 35 to 44 yr old and greater among those over 60 yr. Males are affected significantly more often than females. The incidence among agricultural rent collectors, money lenders, unskilled workers, and businessmen is particularly high, and literate people and those with at least a junior high school education are at a greater risk. The rate among daily tobacco chewers is eight times greater than among nonchewers, and the prevalence is associated with the age at which the habit was taken up and with the frequency of chewing. The risk is significantly higher among those exposed to the quid over 8 hr/day and among those who keep the quid during sleep. If the quid is as much as 50 g, the risk of developing oral cancer is approximately 100 times that for nonchewers. A greater incidence was associated with the use of Mainpury tobacco (a mixture of tobacco leaf, lime, finely cut betel nuts, camphor, and cloves) than with Patiwala tobacco (pure sun-cured tobacco). Tobacco smoking was also associated with an increased incidence of oral cancer, particularly when the burning end of the cigarette was held inside the mouth. Regular drinkers of alcohol have a 10 times greater incidence of oral cancer than do nondrinkers, particularly those who consume at least 450 ml/day and those who use whisky. Cross-tabulation of the data indicated a much higher prevalence rate for oral cancer among Mainpuri tobacco chewers in combination with any other factor (age, sex, education, residence, religion, food, smoking, drinking, or occupation). (2 references)

4714 CANCER IN ISRAEL: SOME ETHNIC CONSIDERATIONS. (Eng.) Modan, B. *In: Cancer Genetics.* Edited by H. T. Lynch. (Springfield, IL: Charles C. Thomas): pp. 185-192; 1976.

A study was made of the incidence of cancer at various sites among different ethnic groups in Israel (European-, African-, Asian-, and Israeli-born Jews, and Arabs) to investigate the relative importance of genetic and environmental factors in carcinogenesis. The principal findings of the study include: (a) there are generally higher incidences at most sites for European-born Israelis and US whites than for Asian- or African-born Israelis and US blacks. (b) There is a higher incidence in females of cancer of the colon and rectum among non-European-born Jews and US whites, but an equal sex ratio for cancer of the thyroid. (c) There are minimal interethnic differences in childhood for all tumor categories combined. (d) There are changes in disease pattern according to length of residence in Israel for gastric cancer, malignant melanoma, and childhood leukemia. The most significant factor in most forms of cancer is the environment, with populations of higher socioeconomic status, such as US whites and European-born Jews, being more susceptible to neoplasms at many sites (particularly the breast, corpus uteri, colon, or rectum) than populations of lower status, such as US blacks and African- or Asian-born Jews, who tend to have a greater prevalence of neoplasms at certain other sites (uterine cervix). (30 references)

4715 STATISTICAL ANALYSIS OF MULTIPLE PRIMARY MALIGNANT TUMORS IN OUR OWN SERIES AND IN CASES REVIEWED FROM JAPANESE LITERATURE. (Jpn.) Inuyama, Y. (Dept. Otorhinolaryngology, Sch. Medicine, Keio Univ., Japan); Saito, S.; Ozu, R.; Horiuchi, M.; Asaoka, K.; Nakajima, Y. *J Otorhinolaryngol Soc Jap* 79(2): 189-202; 1976.

Of 1,093 malignant head and neck tumors seen between 1957 and 1975, 23 cases (2.1%) were multiple primary malignant neoplasms. The average age of the patient was 61 yr. Of 203 cases of multiple primary malignant neoplasms reported in the Japanese literature during the same period, the average age of the patient was 60 yr. There was 140 men and 51 women in this group. Regarding the time interval between the first and second tumors, 9 cases (39%) were synchronous and 14 cases (61%) were nonsynchronous in the group of 23 patients, and 64 cases (35.2%) were synchronous and 118 cases (64.8%) were nonsynchronous in the reviewed cases. Of the 23 cases, malignant antral tumors were most frequent (10 patients) followed by laryngeal cancer (6 patients). Of the literature cases, there were 90 maxillary cancers and 71 laryngeal cancers. Cancers of the bilateral maxillary sinus and double cancers of the larynx were the next most frequent. The causes of multiple primary cancer were discussed. (118 references)

4716 INCIDENCE OF MALIGNANT TUMORS IN CHILDHOOD. FREQUENCY DISTRIBUTION IN AN INPATIENT GROUP OF A PEDIATRIC CLINIC. (Ger.) Jacobi, H. (Universitaets-Kinderklinik, D7800 Freiburg, Mathildenstrasse 1, West Germany); Krapp, R. D. *A Kinderheilkd* 20(4): 277-281; 1975.

An 18-yr survey (1957-1974) of pediatric hospital admissions to the Childrens Hospital of Freiburg, West Germany, was made to test the hypothesis that pediatric malignancy is increasing. There were 575 first malignancies among 67,069 patients 1-14 yr of age. The number of leukemias and solid cancers remained relatively constant, averaging about 32 per year, although the total number of admissions for all conditions rose from 2,463 in 1957 to 4,703 in 1974. Two-thirds of the 575 first admissions were leukemias (42%) and CNS tumors (26%). The total number of pediatric tumor admissions increased from 28 in 1957 to 129 in 1974, but these figures include all readmissions. (4 references)

4717 MANTEL-HAENSZEL ANALYSIS OF OXFORD DATA. I. INDEPENDENT EFFECTS OF SEVERAL BIRTH FACTORS INCLUDING FETAL IRRADIATION. (Eng.) Kneale, G. W. (Regional Cancer Registry, Queen Elizabeth Medical Centre, Birmingham B15, 2TH, England); Stewart, A. M. *J Natl Cancer Inst* 56(5): 879-883; 1976.

A Mantel-Haenszel analysis of data from the Oxford Survey of Childhood Cancers was carried out to determine whether an obvious excess of fetal irradiation histories in the case groups was a genuine finding or an artifact due to associations between obstetric radiography and other birth factors of etiologic importance. Another objective was to determine whether four birth factors (social class, maternal age, sibship, and fetal irradiation) influenced the incidence of childhood cancers. All four originally unmatched factors exerted independent effects on reticuloendothelial system neoplasms (leukemias and lymphomas), and all except sibship position exerted independent effects on other cancers. Regarding sibship position, first-born children were at greater risk, but the risk was the same for all later children. (11 references)

4718 PLASMA OESTRADIOL AND URINARY OESTROGEN METABOLITES IN NORMAL JAPANESE AND BRITISH WOMEN. (Eng.) Kumaoka, S.; Abe, O.; Utsunomiya, J.; Bulbrook, R. D.; Hayward, J. L.; Swain, M. C. *In: Host Environment Interactions in the Etiology of Cancer in Man. Proceedings of a Meeting Held at Primosten, Yugoslavia, 27 August-2 September 1972.* International Agency for Res. on Cancer (Lyon, France): pp. 131-135; 1973.

Premenopausal, menopausal, and postmenopausal women in Japan and Britain were investigated to compare their plasma and urinary estrogens. A single blood specimen was taken on the afternoon of day 1. On days 2-4, three consecutive 24-hr specimens were collected. In premenopausal women, all specimens were collected in the luteal phase of the menstrual cycle; there were no major differences in the amounts of estrone, estradiol, or estriol excreted. In two Japanese and one British woman, urinary pregnanediol and plasma progesterone assays indicated an anovulatory cycle; in the remaining women the mean plasma levels of estradiol were almost identical. There was a pronounced decrease in all estrogen fractions excreted by both Japanese and British menopausal women; the most marked decrease was in the estriol fraction. The differences in plasma estradiol levels between menopausal and postmenopausal women were negligible. The evidence suggests that differences in the estriol ratio between Japanese and Western women aged 15-19 are small and that there may be no significant difference in women aged 35-39 yr. Therefore, if estriol exerts a protective effect against breast cancer, it must do so at an early age. The preliminary data lend no support to the concept that estradiol is directly carcinogenic in women. (12 references)

4719 EFFECT OF AGE ON GROWTH PATTERN. (Eng.) Stoll, B. A. *In: Risk Factors in Breast Cancer*. Edited by B. A. Stoll. (Chicago: William Heinemann Medical Books): New Aspects of Breast Cancer, vol. 2, pp. 129-148; 1976.

There is a common, unproved belief that breast cancer in women varies according to age group, being more malignant in women under 35 yr and less malignant in those over 70 than in the intermediate age group. In both young and older patients, however, there appears to be an almost infinite range of malignant behavior. In Western women, the incidence of breast cancer increases with age, whereas among the populations of Asia, Africa, and Latin America the overall incidence is low and is associated with a risk increasing only until the age of 45, after which it levels or may show a drop. In general, no clear evidence of either a poorer survival or a more advanced presentation of the tumor has been found for younger women. The duration of recurrence-free periods after primary treatment does appear to be lower for women under 40 yr and for those over 70 yr. Changes in the tumor-host relationship at specific times, which can lead to retardation or acceleration of tumor growth, are reviewed critically and related to endocrinological and presumed immunological factors. In a study of 764 patients, the rapid involvement of two or more of the three major localizations of metastases (regional, osseous, and visceral) (termed a ''shower of metastases'') followed by death within 18 mo of first metastasis occurred in 62% of women under the age of 40 at the time of treatment. The proportion decreased steadily with each decade, so that in women over 70 yr, the proportion was only 14%. This difference between the age groups is statistically significant. This sudden failure of host defense is not confined to the premenopausal group and probably reflects immunological rather than endocrinological factors. Tumor doubling time was reported in one study to be shorter in women under the age of 60, but it cannot be assumed that all the clones of a tumor have a similar capacity to establish or maintain metastases. (60 references)

4720 PSYCHOSOMATIC FACTORS AND TUMOUR GROWTH. (Eng.) Stoll, B. A. *In: Risk Factors in Breast Cancer*. Edited by B. A. Stoll. (Chicago, William Heinemann Medical Books): New Aspects Of Breast Cancer, vol. 2, pp. 193-203; 1976.

There have been a number of recent reports suggesting that emotional stress in women may influence the development and growth of breast cancer. The hypothalamopituitary pathway could mediate a cerebral influence on growth. The growth pattern of breast cancer shows a remarkable diversity and is thought to result from interaction between the aggressive potential of the tumor cells and host factors, which may well include psychosomatic factors. Prolactin, growth hormone, and gonadotropin levels can be increased markedly by emotional as well as somatic stress. A higher corticosteroid production has been noted in breast cancer patients who are apprehensive or depressed. An indirect measure of emotional stress derived from the patient's use of tranquilizers, antidepressants, sedatives, or hypnotics was made in a series of 250 patients. Their drug usage in the 12 mo prior to diagnosis was similar to that of women in the general population. However, after diagnosis, the overall drug usage was two to three times higher than in women in the general population. Of five groups of patients distinguished, those with evidence of metastases beyond the axilla at the time of diagnosis and those showing evidence of recurrence or metastases within 12 mo of radical treatment included the highest proportion of those who, in the past, required medication for emotional distress. If a relationship between the persistent emotional stress and an increased rate of breast cancer growth should be confirmed, it does not follow that it is one of cause and effect; they both may arise from a common basis. (49 references)

4721 FACTORS PROVOKING MALE BREAST CANCER. (Eng.) Scheike, O. *In: Risk Factors in Breast Cancer*. Edited by B. A. Stoll. (Chicago: William Heinemann Medical Books): New Aspects of Breast Cancer, vol. 2, pp. 173-192; 1976.

One of the factors that place a male at risk to breast cancer is the Klinefelter syndrome. In this syndrome the gonadotropin excretion is characteristically increased. The possible association between gynecomastia and breast cancer has been reported with a variable incidence ranging from 5% to 19%. After surgery in one group of 79 patients, coexisting gynecomastia was found upon histological examination in 21 cases (27%). There are very little data so far to support the role of estrogen in the induction of male breast cancer. Men with breast cancer excrete a significantly lower amount of pituitary gonadotropins, but otherwise their urinary excretion of hormones does not differ from controls. Heredity studies indicate a relationship between the familial incidence of male and female breast cancer. A comparison of the 5-yr corrected survival rates in a series of men and women revealed that the prognosis in men is not much worse than that in women. The duration of symptoms, the clinical staging at presentation, and the histological grade of malignancy influence the prognosis considerably. No evidence has been found to favor radical mastectomy rather than simple mastectomy in the treatment of male breast cancer. (63 references)

4722 CARCINOMA OF THE PENIS. (Eng.) Persky, L. (Case Western Reserve Univ. Hosp., Cleveland, OH); deKernion, J. *Ca* 26(3): 130-142; 1976.

The current management of penile tumors, with an emphasis on major controversies, is presented, and the treatment of squamous carcinoma is discussed in detail. Most premalignant penile lesions should be completely excised locally. Giant condyloma frequently cannot be distinguished from fungating carcinoma and usually requires limited penectomy. Cancers other than epidermoid carcinoma are very rare and, except for basal cell carcinoma, have a generally poor prognosis. The prognosis of squamous carcinoma, however, depends on the stage of disease as determined by local invasion and involvement of inguinal nodes. Three-year survivals in 55 patients aged 31 to 85 yr were: Stage I (limited to glans, penis, and/or prepuce), 95%; Stage II (shaft or corpora invasion, without nodal or distant metastases), 67%; Stage III (confined to shaft, with proved regional node metastases), 29%; Stage IV (invasion from the shaft, inoperable regional node involvement, or distant metastases), 0%. Most primary lesions were treated by partial penectomy, and no patient developed local recurrence. There was a significant discrepancy between initial clinical and histologic staging due to the difficulty in determining node metastases. To detect and eliminate occult metastases, an aggressive approach to the management of inguinal nodes is proposed, including en bloc ileoinguinal dissection for persistent clinical adenopathy and routine superficial inguinal node biopsy of nonpalpable nodes when the primary invades the corpora. The recently introduced technique of skin bridge node dissection has several important advantages: the groin crease is not incised and the blood supply to the flaps is preserved. (51 references)

4723 SPONTANEOUS TUMORS AND COMMON DISEASES IN TWO COLONIES OF SYRIAN HAMSTERS. I. INCIDENCE AND SITES. (Eng.) Pour, P. (Eppley Inst. for

Res. in Cancer and Allied Diseases, Univ. Nebraska Medical Center, 42d St. and Dewey Ave., Omaha, NB 68105); Kmoch, N.; Greiser, E.; Mohr, U.; Althoff, J.; Cardesa, A. *J Natl Cancer Inst* 56(5): 931-935; 1976.

Reported variations in the frequency, distribution, latency, and morphology of neoplasms among different hamster colonies may be due to a wide range of factors, and as a result the interpretation and comparison of experimental results may not be entirely accurate. To investigate these factors, the occurrence of spontaneous neoplastic and nonneoplastic disease in two colonies of Syrian hamsters was studied. Outbred Syrian hamsters from the Eppley colony (EC; 252 males and 248 females) and from the Hannover colony (HC; 166 males and 211 females) were kept for life, under standard laboratory conditions, in plastic cages in groups of 5 according to sex. They were checked daily and weighed every 2 wk; moribund animals were killed. Uniform methods were used for autopsy and sectioning. Statistical data concerning survival rates and the incidence and organ distribution of disease are given. Significant differences were found in tumor incidence (30%, EC; 42%, HC), occurrence of malignant neoplasms (15%, EC; 32%, HC), appearance of multiple primary tumors in different organs (42%, EC; 30%, HC), average survival rates of tumor-bearing animals, organ distribution and histologic types, and average survival rates of all animals. The frequencies of nonneoplastic diseases also varied between the two colonies. Therefore, precise histologic evaluation is necessary to generate information for comparative purposes when hamsters are used in experimental pathology and carcinogenesis studies. (24 references)

4724 DETERMINANTS OF MORTALITY IN AN INDUSTRIAL POPULATION. (Eng.) Ott, M. G. (Corporate Medical Dept., Dow Chemical Co., 2030 Dow Center, Midland, MI 48640); Holder, B. B.; Langner, R. R. *J Occup Med* 18(3): 171-177; 1976.

The mortality experience of an employee cohort selected from one location of a major chemical manufacturing company (Dow Chemical Co.) was examined to develop background data on an industrial population with emphasis on preselection factors, socioeconomic status, and general job categories. Employees covered by the study comprised 8,171 males (18-64, nearly all of whom were white) from research departments, the corporate headquarters, and a major manufacturing division. Three socioeconomic categories were used in the analysis: (a) managerial, professional, and business personnel and department level supervisors; (b) first line supervisors and employees in skilled trades; and (c) clerical and semiskilled operators and unskilled janitors, packagers, handymen, etc. Differences in mortality were also examined across 15 job categories. Tabulation of expected deaths was based on cause- and age-specific (5-yr age intervals) US white male mortality rates for 5-yr periods from 1952 to 1971. Overall mortality was lower than expected. Statistically significant differences in overall mortality were observed between socioeconomic levels and for each of three cause-of-death categories: cardiovascular disease, malignant neoplasms, and all other causes. Deaths due to malignancies were increased for plant mechanics, machinists, and machine repairmen (28 observed vs 15.8 expected). A complicating factor is that nine deaths, five due to malignancies, occurred among individuals with limited arsenic exposure. Deaths due to malignant neoplasms were greater than expected among welders and organic chemical production area groups. No plausible explanation has been found for the less-than-expected malignancy rate seen among inorganic chemical production workers. (11 references)

4725 PULMONARY CHANGES AMONG VINYL CHLORIDE POLYMERIZATION WORKERS. (Eng.) Lilis, R. (Mount Sinai Sch. Medicine, New York, NY); Anderson, H.; Miller, A.; Selikoff, I. J. *Chest* 69(2): 299-303; 1976.

Pulmonary changes uncovered during clinical examinations of three groups of exposed vinyl chloride (VC) polymerization workers are discussed. The examination included chest x-rays, smoking history, a chronic bronchitis questionnaire, and pulmonary function tests. The first group was from a plant known to have high exposure levels to VC and polyvinyl chloride (PVC). The second plant was the first PVC polymerization facility, so that long exposure effects could be expected. The third plant had a relatively low exposure level. In the first group, the overall prevalence of small linear reticular and/or rounded opacities was 22.7%; x-ray changes increased with length of exposure. In the second group the prevalence of chest x-ray changes was 19.4%. The third group had a much lower prevalence of chest x-ray abnormalities (4.3%). The prevalence of positive smoking history was higher in workers with abnormal chest x-ray films in the first two groups, perhaps indicating a multiple factor effect of smoking and VC-PVC exposure. No significant differences were found between the groups with abnormal and normal chest x-rays with respect to chronic bronchitis or age. Pulmonary function tests showed a relatively high prevalence of obstructive changes, but there was no consistent correlation between abnormal chest x-ray and pulmonary function abnormalities. It is possible that chest x-ray changes and obstructive pulmonary function abnormalities reflect different pathologic processes, the chest x-ray changes being mainly related to parenchymal damage, while the obstructive pulmonary function changes reflect airway changes. (4 references)

4726 IN VITRO AUTORADIOGRAPHIC DETERMINATION OF CELL KINETIC PARAMETERS IN ADENOCARCINOMAS AND ADJACENT HEALTHY MUCOSA OF THE HUMAN COLON AND RECTUM. (Eng.) Bleiberg, H. (Dept. Internal Medicine, Institut Jules Bordet, 1 rue Heger-Bordet, 1000 Brussels); Galand, P. *Cancer Res* 36(2): 325-328; 1976.

Using in vitro double labeling with two different doses of [^{3}H]thymidine, the S-phase duration and the labeling index in biopsy samples taken from human rectal and colonic adenocarcinomas was measured.

Samples from the adjacent healthy mucosa, taken at the same time as the tumor samples, were simultaneously subjected to the same measurements. The mean labeling index in tumors was higher than in the normal mucosa (32.5 and 17.0%, respectively). The mean S-phase value was also longer than in the normal tissue (19.4 versus 11.2 hr). Similar observations were made in tumors of the human epidermis. An increased S-phase duration may be correlated with carcinogenesis. While no definitive conclusions may be reached, an S-phase duration that is longer than normal should be considered a justification for attentive follow-up of the patient.

4727 PARAGANGLIOMAS OF THE HEAD AND NECK (Meeting Abstract). (Eng.) Hamilton, D. G. (Sch. Pathology, Univ. Cape Town, South Africa); Uys, C. J.; Lipper, S. *S Afr Med J* 50(26): 1005-1006; 1976.

4728 NAGASAKI ATOMIC BOMB DOSES (Meeting Abstract). (Eng.) Takeshita, K. (Hiroshima Univ., Hiroshima, Japan). *J Radiat Res* 17(1): 6; 1976.

4729 MORTALITY EXPERIENCE OF A-BOMB SURVIVORS, 1950-1974 (Meeting Abstract). (Eng.) Beebe, G. W. (Radiation Effects Res. Foundation, Hiroshima 730, Japan); Kato, H.; Land, C. E. *Radiat Res* 67(3): 555; 1976.

4730 MORTALITY AMONG A-BOMB SURVIVORS 1950-74 (Meeting Abstract). (Eng.) Kato, H. (Radiation Effects Res. Foundation, Hiroshima, Japan); Beebe, G. W.; Land, C. E. *J Radiat Res (Tokyo)* 17(1): 51; 1976.

4731 PLUTONIUM CONCENTRATION IN HUMAN ORGAN (Meeting Abstract). (Eng.) Takizawa, Y. (Akita Univ., Akita, Japan). *J Radiat Res* 17(1): 5; 1976.

4732 FREEZE-FRACTURE STUDIES ON THE SURFACE MICROVILLI OF SARCOMA 180 ASCITES TUMOR CELLS (Meeting Abstract) (Eng.) Pollock, E. G. (Dept. Biology, California State Univ., Northridge, CA 91324); Kallenbach, R. J. *J Cell Biol* 70(2/Part 2): 274a; 1976.

4733 CELL CYCLE ANALYSIS OF YOSHIDA ASCITES SARCOMA GROWN AFTER A SINGLE CELL TRANSPLANTATION (Meeting Abstract). (Eng.) Ashihara, T. (Dept. Pathology, Shiga Medical Coll., Shiga 524, Kyoto Prefectural Univ. Medicine, Kyoto, Japan); Fujita, S. *J Cell Biol* 70(2/Part 2): 236a; 1976.

See also:

* (Rev): 4211, 4214, 4217, 4228, 4233, 4241, 4245, 4247, 4250, 4255, 4256, 4257, 4258, 4259, 4260, 4261, 4285, 4291, 4292, 4293, 4294, 4295, 4296
* (Chem): 4304, 4310, 4324, 4329, 4353, 4356, 4357, 4360, 4365, 4373, 4376
* (Phys): 4379, 4380, 4402, 4419, 4420, 4422, 4431, 4432, 4433
* (Viral): 4511
* (Path): 4617, 4618, 4632, 4634, 4640, 4650, 4654, 4655, 4657, 4695, 4704

4734 NEW APPROACHES FOR STUDYING SURFACE MEMBRANES. (Eng.) Pasternak, C. A.; Strachan, E.; Micklem, K. J.; Duncan, J. *In: Cell Surfaces and Malignancy. Meeting Held in Bethesda, 11-13 September 1974.* Mora, P. T., ed. John E. Fogarty International Center for Advanced Study in Health Sciences; Natl. Insts. Health. (Bethesda, MD): pp. 147-151; 1975.

Two techniques, useful for the study of membrane phenomena, are discussed. Replacement of the quaternary nitrogen in phospholipids by quaternary phosphorus produces compounds readily detectable by ^{31}P nuclear magnetic resonance measurements. Studies are reviewed that show that the phosphonium analog of choline is metabolized by cells as effectively as (Me-^{3}H)choline. In fact, the growth of various cell lines in the presence of phosphonium choline results in 75% replacement of phosphatidylcholine by the phosphonium analog without apparent loss of cellular integrity or function. Therefore, this analog produces minimally perturbed membranes that are easily studied. An attempt is made to label plasma membranes in this manner; the probe is used to study phospholipid asymmetry. The second technique involves changes in transport mechanisms caused by Sendai virus. This virus increases passive diffusion and decreases facilitated uptake. The permeability changes are triggered by attachment of only a few viral particles per cell, indicating considerable cooperativity at the cell surface. This system should prove useful in determining: (a) the relation between passive diffusion and facilitated uptake, (b) permanent changes in membranes resulting from oncogenic transformation, and (c) the molecular mechanisms involved in passive and facilitated diffusion. This model may also prove useful in the study of the increase in passive diffusion that accompanies the establishment of intercellular junctions in cultured cells. (8 references)

4735 ASSOCIATION OF CELL SURFACE RECEPTORS FOR MELANOTROPIN WITH THE GOLGI REGION IN MOUSE MELANOMA CELLS. (Eng.) Varga, J. M. (Dept. Dermatology, Yale Univ. Sch. Medicine, New Haven, CN 06510); Moellmann, G.; Fritsch, P.; Godawska, E.; Lerner, A. B. *Proc Natl Acad Sci USA* 73(2): 559-562; 1976.

The relationship between the Golgi region and receptors for binding beta-melanotropin (beta-melanocyte stimulating hormone) to mouse melanoma cells was investigated. An association was demonstrated by labeling cells with fluorescein isothiocyanate hormone and by locating the Golgi complex with a histochemical test for thiamine pyrophosphatase activity. The biologically active fluorescent hormone appeared on the surface and later in vesicles in the melanized cells, as judged by fluorescent microscopy. Internalization of the hormone may be instrumental in the process of hormonally induced melanization. Because initial and late events of hormonally induced pigmentation are related to the Golgi complex, it is likely that instructions that follow the attachment of melanotropin to receptors are carried out in a compartmentalized manner.

4736 TRANSPORT OF AMINO ACIDS IN INTACT 3T3 AND SV3T3 CELLS. BINDING ACTIVITY FOR LEUCINE IN MEMBRANE PREPARATIONS OF EHRLICH ASCITES TUMOR CELLS. (Eng.) Cecchini, G. (Dept. Biological Chemistry, Univ. Michigan, Ann Arbor, MI); Lee. M.; Oxender, D. L. *J Supramol Struct* 4(4): 441(401)-447(407); 1976.

To examine amino acid transport and to determine whether transformation results in alteration of transport capability, the transport of amino acids into 3T3 and SV3T3 (SV40 virus-transformed 3T3) cells was measured on glass coverslips. The 3T3 and SV3T3 cells contained both A (alanine preferring) and L(leucine preferring) systems for neutral amino acid transport. Initial rates of uptake of amino acids were about two times higher in SV3T3 than in 3T3 cells. The effect of inhibitors, preloading, and cell density, however, did not indicate marked differences in transport by the two cell types. L-system amino acids, such as leucine, were subject to transstimulation in both cell lines, whereas A-system amino acids, such as alanine and glycine, were not. Leucine was transported to higher levels in confluent cells than in nonconfluent cells. Glycine, however, showed distinctly less transport activity as the cells became confluent. Ehrlich ascites cell plasma membranes were prepared and assayed for amino acid-binding activity. Leucine-binding activity was detected by equilibrium dialysis in Triton X-100-treated membrane preparations. The activity was found only in the presence of detergents, although it does not appear to be completely solubilized. Its specificity is similar to that observed for the transport system in 3T3 and Ehrlich cells, and it is saturable with high concentrations of leucine.

4737 ASYMMETRIC DISTRIBUTION AND NEAREST NEIGHBOR ANALYSIS OF PHOSPHOLIPIDS IN THE ERYTHROCYTE MEMBRANE. (Eng.) Marinetti, G. V. *In: Cell Surfaces and Malignancy. Meeting Held in Bethesda, 11-13 September 1974.* Mora, P. T., ed. John E. Fogarty International Center for Advanced Study in Health Sciences; Natl. Insts. Health. (Bethesda, MD): pp. 49-52; 1975.

Various penetrating and nonpenetrating compounds have been used to study the asymmetric distribution of proteins and phospholipids across cell membranes. The majority of these studies utilized the erythrocyte. The inner surface of the erythrocyte membrane has been found to contain most of the phosphatidylethanolamine (PE) and phosphatidylserine (PS). Studies using 2,3,5-trinitrobenzenesulfate (TNBS, a nonpenetrating probe) and 1,5-difluoro-2,4-dinitrobenzene (a penetrating probe) led to the conclusion that only 8% of the total membrane PE and almost none of the membrane PS were on the outer surface of the erythrocyte membrane. Nearest-neighbor analysis using nonsaturating amounts of difluorodinitrobenzene suggested that 33% of PS is closely associated with membrane proteins, whereas 87% of PE is associated with either other PE molecules or PS molecules. The interaction of certain proteins with PS may be an important step in a nucleation process that may play a role in membrane biogenesis. (10 references)

4738 IN VITRO DERIVED MOUSE A9 CELL CLONES DIFFERING IN MALIGNANCY: ANALYSIS BY SOMATIC CELL HYBRIDIZATION WITH YACIR LYMPHOMA CELL CLONES. (Eng.) Clements, G. B. (Dept. Pathology Univ. Glasgow, Western Infirmary, Glasgow, Scotland); Fenyo, E. M.; Klein, G. *Proc Natl Acad Sci USA* 73(6): 2004-2007; 1976.

Evidence for the simultaneous presence of cells of both high and low malignancy within a single cultured cell line is presented. A culture of mouse A9 cells cloned either by suspension in a 0.3% agarose medium at a density of 10 cells/ml, or in liquid culture with an inoculum of 5 cells/60-mm petri dish yielded clones of both high and low malignancy. Confirmation of these differences was obtained after their hybridization with subclones of YACIR, a lymphoma induced by Moloney leukemia virus. A9 clones of low malignancy suppressed the malignancy of the YACIR partner in the hybrids. Hybrids between a highly malignant A9 clone and two different clones of YACIR complemented to yield hybrids of reduced malignancy. The observations are consistent with the hypothesis that malignancy results from alteration of the genetic information of the cell. No correlation was found between the malignancy of a particular A9 clone and its ability to produce a focus-forming mouse type C virus. (27 references)

4739 SYNTHESIS AND HALF-LIFE OF A SERUM FACTOR IN NORMAL AND TUMOR-BEARING ANIMALS. (Eng.) Dolan, M. L. (Univ. Pittsburgh Sch. Medicine, Pittsburgh, PA 15261); Sudbury, G.; Ove, P. *J Natl Cancer Inst* 56(6): 1261-1267; 1976.

The synthesis and disappearance of a serum factor from the circulating blood of rats bearing Morris hepatomas 7777 or 7800 were described. Evidence was provided that the factor was synthesized by normal rat livers and continued to be synthesized in tumor-bearing animals. Despite very low levels in the sera of tumor-bearing animals, the factor was synthesized at twice the rate in these animals compared to normal rats. The half-life of serum protein factor in circulating blood of normal and hepatoma-bearing animals was also compared. The half-life of the total serum protein was 2 days in normal rats and 2.9 days in hepatoma 7777-bearing animals. The half-life of the serum factor, on the other hand, was 23 hr in normal rats but only 9 hr in hepatoma-bearing rats. This rapid disappearance of the factor from the circulating blood, despite an increased synthesis, accounts for its complete absence in the sera of hepatoma-bearing animals with tumor wt larger than the liver wt. (26 references)

4740 CELL DETACHMENT IN OUABAIN-SENSITIVE AND RESISTANT EHRLICH ASCITES CELLS. (Eng.) Weiss, L. (Dept. Experimental Pathology, Roswell Park Memorial Inst., Buffalo, NY 14263); Mayhew, E.; Huber, D. *Exp Cell Res* 96(2): 388-392; 1975.

The in vitro detachment of Ehrlich-Lettre hyperdiploid ascites carcinoma (EAT) cells from protein-coated glass surfaces was examined in relation to inherited functional changes in their peripheries associated with ouabain resistance. EAT cells resistant (OR) to ouabain, a specific inhibitor of plasma membrane (Na$^+$, K$^+$)-inactivated ATPase, were induced from wild-type ouabain-sensitive (OS) cells. OS cells were more resistant to detachment from substrate than OR cells in the absence of ouabain; treatment with ouabain at 10^{-3} and 10^{-4} M facilitated OS cell detachment. In the presence of high potassium levels, this action of ouabain was partially but not completely reversed. Changes in the (Na$^+$, K$^+$)-ATPase itself and/or other associated membrane changes, rather than K$^+$ transport, are directly or indirectly responsible for the altered detachment patterns. Since the detachment of OS cells is facilitated in ouabain, OS (Na$^+$, K$^+$)-ATPase may be involved in maintaining adhesive stability. The suggestion that protein synthesis may be necessary for the formation of antiadhesion factors is supported. No differences in the release of acid proteases or acid phosphatases were detected between untreated or ouabain-treated OS or OR cells; thus, differences in detachment behavior are not related to lysosomal activation. Results agree with the hypothesis that specificity in cell detachment as well as attachment should be considered in any mechanistic interpretation of cell interactions. (23 references)

4741 PROGNOSTIC SIGNIFICANCE OF INAPPROPRIATE PRODUCTION OF PREGNANCY PROTEINS BY BREAST CANCERS. (Eng.) Horne, C. H. (Dept. Pathology, Univ. Medical Buildings, Foresterhill, Aberdeen AB9 2ZD, Scotland); Reid, I. N.; Milne, G. D. *Lancet* 2(7980): 279-282; 1976.

Fifty cases of breast cancer (diagnosed over a 4-yr period) were studied retrospectively. An enzyme-bridge immunoperoxidase technique demonstrated that so-called pregnancy-specific proteins were detectable in the cytoplasm of the tumor cells. Pregnancy-specific β_1-glycoprotein was present in 76% of cases, placental lactogen in 82%, and chorionic gonadotrophin in 60%. Women with cancers negative for pregnancy-specific β_1-glycoprotein and placental lactogen had a significantly longer survival time compared with those whose cancers stained for these proteins. Pregnancy-specific β_1-glycoprotein appeared to be the best indicator of prognosis. (12 references)

4742 THE INFLUENCE OF THE PROGESTOGEN GESTONORONE CAPROATE ON TESTOSTERONE TURNOVER IN RENAL CELL CARCINOMA. (Eng.) Marberger, M., Jr. (Dept. Urology, Univ. Mainz Medical Sch., Mainz, West Germany); Altwein, J. E.; Orestano, F. *Invest Urol* 13(4): 302-306; 1976.

To investigate the effect of gestagens on renal cell carcinoma 300-mg slices of normal, human kidney, renal cell carcinoma (RCC), and preoperatively irradiated RCC were subjected to a short term incubation with 200 pmoles of ^{3}H-testosterone and 1 and 2 µg of gestonorone caproate. In the normal kidney 61.4% of the testosterone added was metabolized, the oxidation products androstenedione and epitestosterone outweighing by 6:1 the reduction products 5-alpha-dihydrotestosterone and androstanediol. In RCC only

22% of the testosterone was metabolized, with 8.5% being converted to 5-alpha-androstanes. Gestonorone caproate essentially did not influence testosterone turnover. This can be explained by its action as an inhibitor of the reductive pathway of the testosterone metabolism only, which is insignificant in these tissues.

4743 ORGAN CULTURE OF RODENT PROSTATE: EFFECTS OF POLYAMINES AND TESTOSTERONE.

(Eng.) Edwards, W. D. (Dept. Pharmacology, West Virginia Univ. Medical Center, Morgantown, WV 26506); Bates, R. R.; Yuspa, S. H. *Invest Urol* 14(1): 1-5; 1976.

BALB/c mice prostate organs were maintained in culture for 14 days on a plasma clot to which insulin, testosterone, spermine, and spermidine were added. Explants cultured in the control medium (embryo extract and chicken plasma) showed regressive changes, and there was complete necrosis by the fifth day. When cultured in the presence of testosterone plus the control medium, the explants continued to maintain their epithelial height and stromal characteristics after 5 days. By the seventh day regressive changes similar to those seen for explants cultured in the absence of testosterone were observed. With spermine and spermidine in the medium, along with insulin and testosterone, the stromal and epithelial integrity of the explants remained intact for 14 days. When either testosterone or spermine was omitted from this list of additives, survival and maintenance of normal morphology were greatly impaired. Omission of spermidine did not affect results if the other chemicals were added. No attempts were made to maintain cultures beyond 14 days. (23 references)

4744 ELECTRON MICROSCOPY OF DNA CROSSLINKED WITH TRIMETHYLPSORALEN: TEST OF THE SECONDARY STRUCTURE OF EUKARYOTIC INVERTED REPEAT SEQUENCES.

(Eng.) Cech, T. R. (Dept. Biology, Massachusetts Inst. Technology, Cambridge, MA 02139); Pardue, M. L. *Proc Natl Acad Sci USA* 73(8): 2644-2648; 1976.

To investigate the relative frequency of the linear and cruciform forms of palindrome sequences in eukaryotic genomes, the DNA of intact mouse tissue culture cells was covalently cross-linked with 4,5',8-trimethylpsoralen (me$_3$-psoralen) to prevent rearrangement of the secondary structure during DNA isolation. The distribution of me$_3$-psoralen cross-links was determined by electron microscopy after denaturation. The high frequency and the relatively uniform distribution of the me$_3$-psoralen cross-links implied that almost all of the inverted repeat sequences had been cross-linked. In spite of this, no significant number of cruciforms was detected by electron microscopy. To determine whether the me$_3$-psoralen might itself be disrupting cruciform structures, cruciforms were first produced in isolated *Tetrahymena* rDNA by heat treatment and then cross-linked in vitro. The cross-linking stabilized rather than disrupted these cruciforms. It is concluded that the inverted repeat sequences of the mouse tissue culture cells are predominantly in linear forms rather than in cruciform structures inside the cell. (31 references)

4745 TEMPORAL RELATIONS OF CHROMATIN PROTEIN SYNTHESIS, DNA SYNTHESIS, AND ASSEMBLY OF DEOXYRIBONUCLEOPROTEIN.

(Eng.) Seale, R. L. (Div. Molecular and Cellular Biology, Natl. Jewish Hosp. and Res. Center, 3800 E. Colfax Ave., Denver, CO 80206). *Proc Natl Acad Sci USA* 73(7): 2270-2274; 1976.

Chromatin assembly was investigated in terms of the sites on DNA where newly synthesized chromatin proteins associate. Chromatin from HeLa S-3 cells labeled with [^{14}C]-bromodeoxyuridine (BUdR) and [^{3}H]-lysine was fixed with formaldehyde and resolved in CsCl gradients. By variation of the spacing of the labeling intervals of the two isotopes to encompass all possible periods in the S phase, the association of labeled, newly synthesized proteins on newly synthesized (BUdR-substituted) or preexisting chromatin DNA was determined. In all experiments newly synthesized chromatin proteins were predominantly associated with nonreplicating DNA. Possible mechanisms by which cells recycle preexisting chromatin proteins to restore the protein content of newly synthesized DNA are discussed. (38 references)

4746 TISSUE-SPECIFIC INHIBITION OF DNA SYNTHESIS BY LUNG AND LIVER Ca-FREE SALT EXTRACTS ABLE TO INCREASE MUTUAL CELL-TISSUE ADHESION.

(Rus.) Modianova, A. A. (Inst. Experimental and Clinical Oncology, Moscow, USSR); Malenkov, A. G. *Tsitologiia* 17(10): 1155-1159; 1975.

The effect on DNA synthesis of Ca-free salt extracts with adhesive activity obtained from the lungs and liver of adult mice, rats, and smelts was studied. The extracts caused reversible inhibition of DNA synthesis in the tissue from which they were obtained; i.e., their action was tissue-specific rather than species-specific. The inhibition of DNA synthesis by the extracts may be mediated through their stabilizing effect on cell membranes. The tissue-specific adhesive factor may play the role of G$_1$-chalone in epithelial tissue.

4747 INCREASING CONTENT OF POLY A(+) tRNA OF SERUM-STIMULATED CELLS IN THE ABSENCE OF RIBOSOME SYNTHESIS.

(Eng.) Johnson, L. F. (Dept. Biology, Massachusetts Inst. Technology, Cambridge, MA 02139); Penman, S.; Green, H. *J Cell Physiol* 87(2): 141-146; 1976.

When 3T6 cells undergo a serum-induced transition from resting to growing state, the number of ribosomes and the amounts of mRNA and tRNA increase as the cells prepare for DNA synthesis. The effect of preventing ribosome synthesis during this transition was examined. When resting cells were stimulated to grow in the presence of 5-fluorouridine, mRNA accumulated normally during the first eight hr, though new ribosome formation was completely blocked by the drug. At later times, mRNA continued to accumulate, but at a reduced rate. The ratio of poly A(+) mRNA to rRNA increased from the value characteristic of resting 3T6 (1.8%) to that of growing 3T6 (2.7%) by 5 hr, and continued to increase to abnormally high values after this time. Although labeling of tRNA was not affected after brief exposure of cells to fluorouridine, the drug prevented the later accumulation of tRNA that ordinarily occurred following serum stimulation of resting cells. This failure of accumulation was not the result of increased lability of fluorinated tRNA, but was probably due to failure of the transcription rate of pre-tRNA to increase. It is possible that this effect might be due to a regulatory system coupling tRNA content to ribosome content. In cultures stimulated with serum in the presence of fluorouridine the rate of protein synthesis increased with poly A(+) mRNA content during the first 8 hr; it then failed to increase further, possibly because ribosomes become rate-limiting.

4748 NUCLEAR PROTEIN KINASES FROM MURINE CELLS. (Eng.) Schlepper, J. (Fachbereich Biologie der Universitaet Konstanz, D-7750 Konstanz, Postfach 7733, West Germany); Knippers, R. *Eur J Biochem* 60(1): 209-220; 1975.

Three protein kinase activities were found in nuclei from three rapidly proliferating homogeneous cell populations: (a) mouse Ehrlich ascites tumor cells kept ip in inbred BALB mice and harvested 6-7 days after inoculation, (b) mouse L cells (L929) cultivated in Dulbecco's modified Eagle medium with 10% calf serum, and (c) rat C6 glioma cells cultivated in Ham's F medium with 10% calf serum. Two of the activities were soluble, whereas one was bound to chromatin. Similar chromatographic patterns were observed for each of the cell types tested. Both of the soluble enzymes depended on cyclic AMP (optimal concentration 5 μM), but the activity of the chromatin-bound enzyme was not affected by cyclic AMP. The soluble protein kinases were composed of at least two subunits and were similar, if not identical, to the corresponding cytoplasmic protein kinases. The chromatin-bound kinase was probably composed of one polypeptide chain with an estimated molecular wt of 80,000-90,000. The chromatin-bound enzyme required higher magnesium salt concentrations for enzymatic activity and was less sensitive to an increase in the concentration of monovalent cations than the soluble enzymes. The chromatin-bound kinase also differed from the soluble kinases in that it appeared to be located only in the nucleus. This chromatin-bound enzyme may be analogous to the nuclear cyclic AMP-dependent protein-kinase K II of Chinese hamster cells. (38 references)

4749 PYRUVATE KINASE ISOZYMES IN VARIOUS TISSUES OF RAT, AND INCREASE OF SPLEEN-TYPE PYRUVATE KINASE IN LIVER BY INJECTING CHROMATINS FROM SPLEEN AND TUMOR. (Eng.) Muroya, N. (Inst. for Protein Res., Osaka Univ., Suita, Osaka 565, Japan); Nagao, Y.; Miyazaki, K.; Nishikawa, K.; Horio, T. *J Biochem* 79(1): 203-215; 1976.

Pyruvate kinase in various Donryu rat tissues was separated by the isoelectric method into seven kinds of p*I*-isozymes; some of these p*I*-isozymes contained bound fructose 1,6-diphosphate (FDP). The bound FDP was completely dissociated when the isozymes were salted out with ammonium sulfate. In the FDP-free form, pyruvate kinase was classified into liver type (type L), p*I* 6.2; muscle type (type M), p*I* 7.4; and spleen type (type M_2), p*I* 7.8. Type M had no FDP binding site. Type M_2 had two kinds of FDP binding sites; the FDP was easily dissociable at one site but not at the other. When the ionic strengths of the solutions were low, binding could occur at both sites; at high ionic strength the second site could associate FDP only if the first site had bound FDP. Type L had two kinds of FDP binding sites; the association and dissociation of FDP at both sites was reversible in the presence or absence of KCl. When the Rhodamine sarcoma was transplanted, the content of type M_2 isoenzyme increased in the livers of tumor-bearing rats. When rats were injected with chromatin prepared from either the sarcoma or spleen, the M_2 isoenzyme levels in the livers again increased. Chromatin prepared from liver had no effect on the isoenzyme pattern; this indicated that the factor capable of controlling the gene expression was present in chromatins of sarcoma and spleen but barely or not at all in liver chromatin. (40 references)

4750 STUDY OF THE FACTORS OF POSTTRANSCRIPTIONAL REGULATION OF THE RATE OF CATALASE SYNTHESIS IN THE LIVER OF TUMOR-BEARING RATS. (Rus.) Komov, V. P. (Leningrad Chemical Pharmaceutical Inst., Leningrad, USSR); Rakhmanina, T. F.; Bulat, A. D. *Biull Eksp Biol Med* 81(3): 303-305; 1976.

Total catalase activity in the liver of tumor-bearing rats is markedly reduced compared with that of normal animals. Two cytoplasmic factors, activating (F-act) and inhibiting (F-inh), respectively, are believed to regulate catalase synthesis. Pliss lymphosarcoma was transplanted sc in male randombred rats who were then treated with 3-amino-1,2,4-triazole (1 g/kg, ip) and in 24 hr with F-act and F-inh (0.2, 0.6, and 1.2 ml, iv). The authors concluded that inhibition of catalase synthesis is correlated with the F-act/F-inh ratio, which was reduced on day 2 (1.78), reached a minimum on day 7 (0.57), and was slightly increased on day 14 (1.59). Control value was 3.14. (15 references)

4751 KEY ENZYMES OF IMP METABOLISM: TRANSFORMATION- AND PROLIFERATION-LINKED ALTERATIONS IN GENE EXPRESSION. (Eng.) Weber, G. (Lab. Experimental Oncology, Indiana Univ. Sch. Medicine, Indianapolis, IN); Prajda, N.; Jackson, R. C. *Adv Enzyme Regul* 14: 3-24; 1976.

Enzymes of inosine 5'-phosphate (IMP) metabolism during normal and neoplastic liver proliferation and for a spectrum of hepatomas of different growth rates were examined in inbred strains of male Buffalo and ACI/N rats and male Wistar rats. An effort was made to determine whether the key enzymes of IMP synthesis, degradation, and utilization show characteristic patterns in proliferative conditions. The activities of adenylosuccinate synthetase and amidophosphoribosyltransferase were increased in all hepatomas (regardless of growth rate) by about two- to fourfold, and adenylosuccinase activity was increased 1.2 to twofold over the activity for normal rat liver in all hepatomas. The activities of the catabolic enzymes, xanthine oxidase and uricase, were decreased from normal in all hepatomas by 2 to 10 times. Since the above enzymes showed alterations in activity for all hepatomas, even the slowest growing (12.4 mo), this reprogramming of gene expression appears to be linked with malignant transformation per se. In contrast, IMP dehydrogenase was an indicator of both malignant transformation and growth-rate. IMP activity showed a two- to threefold increase in slow growing hepatomas (12.4 mo) and increased in parallel with tumor growth rates, reaching a 12- to 13-fold increase over its activity in normal rat liver for the most rapidly growing hepatomas (0.6 mo). IMP dehydrogenase was also the earliest enzyme to rise in the regenerating liver (500% increase in specific activity in 6-18 hr). Its increased activity was in the range observed in the newborn rat liver; however, it was markedly lower than that observed in very malignant hepatomas that exhibit the same growth rate as the regenerating liver. The discriminating power of the biochemical pattern of enzymes of purine metabolism indicates that imbalance in the alteration pattern of key enzymes in IMP synthesis, degradation, and utilization is specific to malignancy. The pattern of these enzyme activities also provides quantitative and qualitative information for identifying regenerating and differentiating liver and for distinguishing the enzyme patterns from those observed in normal and rapidly growing neoplastic liver. (21 references)

4752 DIFFERENT ELECTROPHORETIC DISTRIBUTION PROFILES OF HUMAN BONE MARROW STEM CELLS.

(Eng.) Schubert, J. C. F. (Centre Internal Medicine, Dept. Hematology, University Hosp. Frankfurt/Main, D-6 Frankfurt 70, West Germany); Walther, F. *Adv Biosci* 14: 299-308; 1975.

Human bone marrow cells were separated by preparative continuous free-flow electrophoresis, and the fractionated cells were examined with the agar colony unit (ACU) and diffusion chamber techniques. Colony-forming units were studied by a modified semisolid agar colony technique based on a carbon dioxide-free medium buffered with carrier ampholytes, thus stabilizing the pH. Human leukocytes were used as the feeder layer. The ACU distribution of normal bone marrow cells after electrophoretic separation revealed that most of the colonies originated from bone marrow cells with high electrophoretic mobility (EPM); i.e., the region of blast cells and immature granulopoietic cells. Bone marrow cells with low EPM were almost unable to produce colonies. Identical results were obtained with bone marrow cells of chronic myeloid leukemia, apart from the higher colony rate of the latter. The colonies observed were granulocytic-monocytic and were only obtained with cells of the blast cell fractions. In cultures of acute myeloid leukemia, the results were similar; however, two populations of blast cells were distinguished from each other by their different EPMs. The number of colonies, all granulomonocytopoietic, correlated with the blast cell content in three pooled fractions. Application of the diffusion chamber technique to the fractionated cells revealed blast cells, granulocytopoiesis, monocytes, and megakaryopoiesis, but no clearly defined erythroblasts. In contrast to the ACU technique, which revealed high EPM stem cells only, both high and low EPM types were revealed. Those with low EPM showed granulopoiesis, monocytopoiesis, and megakaryopoiesis. The fact that graft-versus-host reactive lymphocytes are marked by a distinct EPM differing from the slowly migrating stem cells may be of clinical importance in grafting allogenic bone marrow cells. (11 references)

4753 MOUSE GLOBIN GENE EXPRESSION IN ERYTHROID AND NON-ERYTHROID TISSUES.

(Eng.) Humphries, S. (Beatson Inst. Cancer Res., Glasgow, G3 6UD, Scotland); Windass, J.; Williamson, R. *Cell* 7(2): 267-277; 1976.

Using cDNA hybridization, mouse globin RNA sequences could be detected in the nuclear and cytoplasmic RNA not only from erythroid cells (fetal liver and reticulocytes), but also in low amounts in non-erythroid tissues (adult brain and liver, and cultured lymphoma, untransformed, and transformed fibroblast cell lines). For the nuclear RNAs, melting curve, density, and size determinations on the hybrids confirmed the presence of an RNA species indistinguishable by cDNA hybridization from reticulocyte globin mRNA. Thus the genes coding for alpha- and beta-globin, proteins thought to be found only in a restricted range of differentiated cell types, may nonetheless be transcribed at very low levels in all mouse cells. The proportion of the globin RNA sequences in the nuclear RNA that are found associated with poly(A) sequences does not vary markedly from one tissue or cell line to another. In erythroid cells, however, the proportion of the cellular globin RNA sequences that are found in the cytoplasm is very much higher than in non-erythroid cells. This suggests the existence of messenger-specific nuclear post-transcriptional control mechanisms which do not use poly(A) as a signal, and which allow the cytoplasmic accumulation of globin mRNA only in erythroid tissues.

4754 PROSTATIC TISSUE CELL GROWTH AND ASSESSMENT.

(Eng.) Merchant, D. J. (Dept. Microbiology and Cell Biology, Eastern Virginia Medical Sch., Post Office Box 1980, Norfolk, VA 23501). *Semin Oncol* 3(2): 131-140; 1976.

The in vitro study of prostatic tumors is discussed. Although the nature of the organ and the lack of basic information about it limit the usefulness of in vitro techniques at present, there are organ culture methods that can yield valuable information. Several important questions must be answered for cell culture methods to achieve their potential: effect of disaggregation procedures on prostatic tissue; appropriate selection of specimens, particularly controls; ability to clearly identify and select cell types; and how to overcome the variable of serum as a source of hormones. Recent developments in the area of cell and organ culture seem most encouraging. Two continuously propagated cell lines of putative prostatic origin have been described. One of these developed in culture apparently as the result of a spontaneous transformation, while the other was propagated from an adenocarcinoma. If these cell lines can be proved to be representative of the tissues of origin, in vitro systems can soon be applied to study of the prostate. (55 references)

4755 SOMATIC CELL HYBRIDIZATION OF MOUSE MYELOMA CELLS. (Eng.) Margulies, D. H. (Dept. Cell Biology, Albert Einstein Coll. Medicine, Bronx, NY 10461); Kuehl, W. M.; Scharff, M. D. *Cell* 8(3): 405-415; 1976.

Somatic cell hybrids between different mouse myeloma cell lines were isolated using modifications of existing techniques. The hybrid nature of these cells was established by HAT (hypoxanthine/amethopterine/thymidine) or HAT-ouabain selective procedures, their chromosome number, and, in one case, H-2 surface antigen expression. Three hybrid lines are described; an IgG$_2$b, κ x Ig$_2$a, κ; an IgG$_1$, κ x IgG$_2$b κ; and an IgG$_1$, κ x IgM, λ. In all cases, both parental types of H and L chains were expressed in the hybrid cells and no new chains were observed. However, molecules possessing disulfide-bonded mixtures of parental H and/or L chains were seen. Analysis of subclones of these hybrids indicated considerable stability in the expression of the immunoglobulins for up to 13 mo. However, segregant clones no longer synthesizing one or more of the parental H or L chains arose frequently. (56 references)

4756 L-LACTATE TRANSPORT IN EHRLICH ASCITES-TUMOR CELLS. (Eng.) Spencer, T. L. (Dept. Physiological Chemistry, Johns Hopkins Univ. Sch. Medicine, 725 North Wolfe St., Baltimore, MD 21205); Lehninger, A. L. *Biochem J* 154(2): 405-414; 1976.

Ehrlich ascites-tumor cells were investigated with regard to their ability to transport L-lactate by measuring either the distribution of [^{14}C]lactate or concomitant H$^+$ ion movements. The movement of lactate was dependent on the pH difference across the cell membrane and was electroneutral, as evidenced by an observed 1:1 exchange for OH-ions or 1:1 co-transport with H$^+$ ions. Kinetic experiments showed that lactate transport was saturable, with an apparent Km of approx 4.68 mM and a Vmax as high as 680 nmol/min per mg of protein at pH 6.2 and 37 C. Lactate transport exhibited a high temperature dependence (activation energy = 139 kJ/mol). Lactate transport was inhibited competitively by (a) a variety of other substituted monocarboxylic acids (e.g. pyruvate, K_1 = 6.3 mM), which were themselves transported, (b) the non-transportable analogues, α-cyano-4-hydroxycinnamate (K_1 = 0.5 mM); α-cyano-3-hydroxycinnamate (K_1 = 2 mM) and DL-p-hydroxyphenyl-lactate (K_1 = 3.6 mM) and (c) the thiol-group reagent mersalyl (K_1 = 125 μM). Transport of simple monocarboxylic acids, including acetate and propionate, was insensitive to these inhibitors. Experiments using saturating amounts of mersalyl as an 'inhibitor stop' allowed measurements of the initial rates of net influx and of net efflux of [^{14}C]lactate. Influx and efflux of lactate were judged to be symmetrical reactions in that they exhibited similar concentration dependence. It is concluded that lactate transport in Ehrlich ascites-tumor cells is mediated by a carrier capable of transporting a number of other substituted monocarboxylic acids, but not unsubstituted short-chain aliphatic acids.

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